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STUDIES IN REFLEXES

HISTORY, PHYSIOLOGY, SYNTHESIS AND NOMENCLATURE: STUDY II

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BRACHIORADIAL REFLEX

This is the most important reflex of the upper extremity. Of particular significance, especially in diagnosis of a transverse lesion in the cervical part of the spinal cord, is the comparative strength of the brachioradial and triceps reflexes. The importance of this point can hardly be overestimated. The brachioradial reflex is elicited by a brisk stretching of the brachioradial muscle, formerly called the supinator longus muscle. Monrad-Krohn [161] described it under the heading "radialis periosteal reflex (supinator jerk)." This reflex is also often called the radius reflex, the forearm periosteal reflex, the radioperiosteal reflex, the supination reflex, the wrist jerk, the forearm reflex, the styloradial reflex, the radioflexor reflex, the radiobicipital reflex and the reflex of the head of the radius. It is hardly necessary to dwell on the technic of its elicitation. This is true of practically any other reflex. If the topography and function of the muscle and the alinement of its fibers are kept in mind, it is simple for any one to bring the forearm into such a position and to administer such a blow in such a direction as to effect exclusively or predominantly a brisk stretching of the brachioradial muscle. The optimal position of the forearm is that of a supination of 45 degrees. When the forearm is in a position of extreme pronation, the reflex is much weaker, and it does not appear at all if the forearm is completely supinated. It is best to tap the lower third of the radius; the processus styloideus radii or the capitulum radii may also be tapped. With such a tap on the bone, which is aimed primarily at the brachioradial muscle, usually not only this muscle but the biceps muscle, as well as the flexors of the fingers and of the hand, often contracts. Even the deltoid, the latissimus dorsi and the pectoralis muscles may participate. But it is not quite correct to regard the finger flexion as "part of the complete radial or supinator reflex," as Wilson [139] did. The finger flexion elicited by tapping the radius is a distinct entity, elicited at the same time as the brachioradial reflex, but in no way "part" of it.

In the light of what has been said previously, it is hardly necessary to emphasize that the brachioradial reflex is not a periosteal reflex. The role of the periosteum here is completely without significance. This has been proved by the experiments of Hoffmann [9] and Dumpert and Flick.[166] The reflex is preserved even though the periosteum as a reflex receptor is completely eliminated by procaine. Hoffmann even assumed that by production of pain stimulation of the periosteum impedes the course of the reflex. Dumpert and Flick, in summarizing their experiments with the brachioradial reflex, made the essential point applicable to all reflexes, in stating:

> The movements caused by the reflexive muscle contractions are always antagonistic to the movements to which the forearm is subjected primarily by the tapping.

This and other physiologic experimental investigation in the last decades proves the myogenic nature of the deep reflexes. The work had a great deal to do with the brachioradial reflex in particular. In view of all this, one is amazed —to say the least—at finding the following statement in a popular textbook on neurology which has gone through several editions: "The radial reflex . . . is a periosteal and not a tendon reflex." This attempt to differentiate between periosteal and tendon reflexes or between bone and tendon reflexes (Bickel [149]) is futile, since all these reflexes are one and the same muscle stretch reflex.

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Dedicated, in deep gratitude, to Dr. Langley Porter, dean of the University of California Medical School, 1927 to 1941.

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166. Dumpert, V., and Flick, K.: Zur Frage der Periostreflexe, J. f. Psychol. u. Neurol. **29**:153, 1923.

ELICITATION OF MULTIPLE REFLEXES

As was evident in the discussion of the pronator and the brachioradial reflex, it is usually difficult—and sometimes well nigh impossible—to elicit only the particular reflex at which one is aiming. To achieve this, and to avoid elicitation of other, unwanted, reflexes, it is necessary to employ a subtle, delicate technic. One must bear in mind that it is not easy on consecutive taps to hit exactly the same spot with exactly the same strength in exactly the same direction. One is apt, therefore, to elicit different combinations of reflexes with different taps. The reflexes which appear with the leading reflex are called synchronous, or concomitant, reflexes. Sternberg [8] spoke of multimuscular reflexes; Austregesilo,[167] of synreflexia, or association of reflexes, and Guillain and Alajouanine,[168] of diffused reflexes. These multiple reflexes, which appear together with, or sometimes even instead of, the reflex which one is trying to elicit, may be divided into three types. These must be kept distinct, for their physiologic mechanisms are entirely different.

1. The first type concerns the concomitant appearance of a skin reflex. Such a reflex may accompany the elicitation of a deep muscle reflex or of another skin reflex. In stimulating the inner side of the thigh, one may elicit not only the cremasteric but the fascia lata reflex. These skin reflexes, appearing with other skin reflexes or with deep muscle reflexes, are clinically insignificant, and their nature is easily discernible.

2. The second, and most important, type of multiple reflexes may arise from the purely mechanical transmission of the concussion. The concussion of the tapped bone affects, of course, all the muscles attached to or traversing this bone and thus brings about the reflexes of these muscles. A tap on the bone in a certain direction may be transmitted to other bones of the extremity according to the position of the bone. What is more important, this concussion may be transmitted through the thorax or through the pelvis to the opposite side and thus produce a contralateral reflex. One must further consider that the reflexive movement of the muscles which one primarily intended to evoke may act by itself on other muscles, directly or indirectly through the bone, and thus evoke their reflexes. It must be emphasized that the muscles affected by one tap do not necessarily belong together functionally, as Böhme [115] assumed, but simply happen to be attached to the tapped bone or to lie within the sphere of the blow. The instances in which such mechanical transmission of a reflex impulse may take place are too numerous to be listed here. Suffice it to mention several outstanding examples. It has just been shown how one may elicit a reflexive movement of various muscles on tapping the radius for elicitation of the pronator or the brachioradial reflex. On tapping the styloid process of the ulna one may achieve contractions of the triceps, the deltoid and even the supraspinati and infraspinati, the rhomboids, the trapezius and the biceps muscle (Myerson [169]). Sternberg [8] pointed out that the tapping of the patellar tendon involves not only the quadriceps muscle but often the adductors and the flexors of the thigh, the tibialis anticus and gastrocnemius muscles and several abdominal muscles. Guillain and Alajouanine [168] offered striking examples of multiple reflexes: contraction of the adductors, the sartorius, the tensor fasciae latae and the gluteus maximus muscle when the dorsum or the planta pedis is tapped; contraction (aside from that of the abdominal and adductor muscles) of the latissimus dorsi and the deltoid muscles when the os pubis is tapped, and contraction of the pectoral and deltoid muscles when the forehead is tapped, as is done for the elicitation of the orbicularis oculi reflex. Foerster and Altenburger [170] often noted involvement of the biceps femoris muscle on tapping for the achilles reflex. The triceps and the adductor muscles of the arm may contract bilaterally when the vertebrae are tapped. Strümpell [171] spoke of a "distant reflex." This distinction is hardly justifiable, since practically any muscle reflex can be elicited from either a near or a distant point. For instance, in order to elicit the reflexes of the semimembranous and semitendinosus muscles, the points of insertion of these muscles at the tuberosity of the tibia or the distant achilles tendon may be tapped. In either case, the reflex elicited is the same; the difference lies only in the length of the lever arm. When the acromion or the medial edge of the scapula is tapped, reflex action is elicited in so many muscles that their individual reaction can hardly be exactly analyzed; the effect of such a tap may extend so far as to include the finger flexors.

167. Austregesilo, A.: Synreflexia (Association of Reflexes), J. Nerv. & Ment. Dis. **68**:1, 1928.

168. Guillain, G., and Alajouanine: Sur la diffusion des réponses au cours de la recherche des réflexes tendineux dans un cas de sclérose diffuse du névraxe, Rev. neurol. **30**:450, 1923.

169. Myerson, A.: Preliminary Paper on Some Unfamiliar and Some New Periosteal Reflexes, Arch. Int. Med. **10**:31 (July) 1912.

170. Foerster, O., and Altenburger, H.: Beiträge zur Physiologie und Pathophysiologie der Sehnen- und Knochenphänomene, Ztschr. f. d. ges. Neurol. u. Psychiat. **147**:779, 1933.

171. Strümpell, A.: Zur Kenntniss der Sehnenreflexe, Deutsches Arch. f. klin. Med. **24**:175, 1879.

On tapping the middle of the upper lip, one may elicit the snout reflex and another reflex, functionally different, the head retraction reflex. On tapping the abdominal muscles, one elicits their deep muscle reflex; at the same time, one jars, at both points at which the abdominal muscles are attached, the pelvis and the thorax, and thus one may also elicit reflexes of the muscles attached to these structures. When one taps the patella of a patient who is in a sitting position or the heel of a patient who is prone, the blow of the reflex hammer can easily be transmitted to the pelvis through the thigh or through the leg respectively. Tapping of the planta pedis influences the plantar muscles, but at the same time it evokes a plantar flexion of the foot by stretching the plantar flexors of the foot.

It is essential to keep in mind that concussion of the bone which takes place when one attempts to elicit a reflex can, under favorable conditions, be mechanically transmitted to the opposite side, the thoracic cage or the pelvis acting as a transmitter. Here, the considerable elasticity of the pelvis and thorax plays a decisive role. The elasticity of the thorax, for instance, is best demonstrated by the fact that on unilateral elicitation of the pectoral muscle reflex, the contralateral reflex appears as well in 15 per cent of cases (Pelliccioli [172]). The reflex phenomenon of Myerson [173] (contraction of the biceps and the pectoralis major muscle on both sides, or on the opposite side only, when the middle of the clavicle is tapped) and the reflex of Chodzko [174] (contraction of several muscles of the upper extremity, unilaterally or bilaterally, when the manubrium sterni is tapped) also show how easy it is to get a bilateral or a contralateral response from muscles attached directly or indirectly to the thoracic cage when this structure is subjected, directly or indirectly, to concussion. It is of historical interest that in a footnote to an article published in 1875 Westphal [175] mentioned that in 1 case he obtained, even on slight tapping of the clavicle, strong contraction of the deltoid, supinator longus and biceps muscles of the opposite side. This contralateral reflex was demonstrated by Strümpell [171] in 1879. Yet, in 1914 Myerson [173] claimed that a contralateral response in elicitation of reflexes "has not hitherto been mentioned." The muscles attached to the pelvis show an especially great susceptibility to indirect concussion. It is remarkable that, as a result of a counterblow, the muscles attached to the opposite side of the pelvis or of the thorax manifest their susceptibility in greater degree than those on the side on which the blow is struck. This phenomenon will be discussed in connection with the adductor reflex of the leg.

3. Of paramount importance for the understanding of the phenomenon of multiple reflexes is another type of multiple reflex. Here the concomitant reflexes do not appear as a result of a direct or an indirect, or distant, concussion and longitudinal stretching of the muscles, but belong to a quite different group—a group which is known by such designations as reflexes of spinal automatism, reflexes of defense, flexor withdrawal reflexes, associated movements and postural reflexes. Marie and Foix [176] distinguished among the phenomena of spinal automatism two chief types of reflexive movements: a flexor and an extensor synergy—*réflexe des raccourcisseurs* and *réflexe des allongeurs*. These two reflexes may alternate and thus produce, for example, the automatism of walking ("stepping reflex" of Sherrington). On elicitation of these reflexes of spinal automatism a great variety of reflexive movements may follow. These responses may be homolateral, contralateral or bilateral. As associated movements they may produce a reflexive action homolaterally, from one extremity to the other or within one extremity. There may be every possible combination of flexor and extensor movements. Especially important here are the crossed flexor reflex and the crossed extensor reflex. It is of further importance to bear in mind that the reflexive phenomena may differ in character, according to the strength of the stimulus, and that the flexor or extensor mechanism brought into play may be incomplete and involve only the outer periphery of the extremity. Thus, dorsiflexion of the big toe may appear in isolated form, to represent the flexor mechanism; plantar flexion of the toes may appear, to represent the extensor mechanism.

Spinal automatism may be brought into play by two kinds of stimuli—interoceptive and exteroceptive. To the first type belongs stretching, even slow stretching, of muscles, often distant ones. To the exteroceptive type belongs any stimulus applied to the skin or that produced by grasping the muscles in any part of the area subjected to spinal automatic control.

172. Pelliccioli, V.: Il riflesso costo pettorale, Rassegna di studi psichiat. **26**:229, 1937.

173. Myerson, A.: Contralateral Periosteal Reflexes of the Arm, J. Nerv. & Ment. Dis. **41**:162, 1914.

174. Chodzko, W.: Sur un nouveau symptôme des lésions organiques du système nerveux central. Signe sternal, Rev. neurol. **65**:131, 1936.

175. Westphal, C.: Ueber einige Bewegungs-Erscheinungen, Arch. f. Psychiat. **5**:803, 1875.

176. Marie, P., and Foix, C.: Les réflexes d'automatisme médullaire, Rev. neurol. **20**:657, 1912.

Thus, tapping the achilles tendon may produce dorsiflexion of the foot instead of plantar flexion. This phenomenon, then, is not a muscle stretch reflex, since the tapping of the achilles tendon acts here not as a stretch stimulus to the triceps surae muscle, but as a banal stimulus to evoke a homolateral flexor withdrawal reflex, of which dorsiflexion of the foot is only a part. Although it may be difficult, or even impossible, in some cases to distinguish between these two types of reflexes, namely, the muscle stretch reflexes and the reflexes of spinal automatism, an effort should nevertheless be made to differentiate between them. Study of the literature reveals that much confusion has been caused by failure carefully to consider this important point.

It must further be taken into account that, in trying to elicit a deep muscle reflex, one may apply a stimulus which, while not causing stretching of any muscle, may evoke a grasp reflex consisting of slow flexion of the fingers or toes. It is well to emphasize that the grasp reflex may be found also in the foot, as demonstrated first by Barraquer [177] and later by Brain and Curran.[178] Usually, it is easy to distinguish the slow grasp reflex from the quick reflexive bending of the fingers or toes due to muscle stretch.

BABINSKI'S INVERSION OF THE RADIAL REFLEX

That a multitude of reflexes can be elicited with a single tap makes it easy to understand certain peculiar reflex phenomena. It has just been shown that tapping of the bone for elicitation of the brachioradial reflex may bring a number of reflexes into play. If there is a lesion in the fifth cervical segment, tapping of the radius will effect only flexion of the hand and fingers, but not flexion of the forearm, or at best an insignificant one, since the fifth cervical segment mediates the latter movement. This phenomenon was first described by Babinski [183] and called "inversion of the radial reflex." Grinker [179] stated: "With a tumor at C 5, percussion of the styloid process of the radius may cause no pronation but flexion of the fingers—inversion of the cubital reflex." This is not quite correct. Here, not the failure of pronation, but the failure of flexion of the forearm is essential. A voluminous literature, as well as the recent article of Walshe and Ross,[180] has confirmed the observation of Babinski. Gendron [181] devoted his doctor's dissertation to this subject. The inversion of the brachioradial reflex is now generally regarded as indicative of a lesion of any kind affecting the fifth and sixth cervical segments of the cord. Yet it must be pointed out that the so-called Babinski inversion of the radial reflex by no means always indicates an organic lesion, as the textbooks would have one believe. There can be no doubt that under special circumstances, and with special technic, one can in normal persons elicit a finger flexor reflex without flexion of the elbow. My experiences in this respect are similar to those of Sahli,[185] Goldstein [148] and Trömner.[112] The last-named author observed Babinski's inversion of the radial reflex in normal and in neurotic persons. In his first communication on this inversion, Babinski [183] emphatically stated that tapping of the radial bone in a normal person never produces flexion of the fingers. This view is not correct. Flexion of the fingers does occur in normal persons without flexion of the forearm. Later Babinski [183] modified his statement by saying that there is sometimes a flexion of the hand and fingers, too, but of lesser strength than that of the forearm.

Further, such an inversion does not necessarily indicate a lesion of the spinal cord but may mean a peripheral lesion. It may be present, for instance, in cases of myopathy, as André-Thomas [184] mentioned. The greatest objection to Babinski's term is its physiologic incorrectness. In this phenomenon no inversion takes place. There is no replacement of one reflex by another, as Walshe [185] interpreted it. The fact is that on tapping the radius one may elicit several entirely separate reflexes. A localized lesion of the spinal cord or of its efferent tracts may lead to the absence of one of these reflexes. If there is a transverse lesion of the spinal cord certain reflexes may be absent, and the reflexes mediated through the lower level of the spinal cord may be exaggerated. But this exaggera-

177. Barraquer, L.: Contribution a l'étude du réflexe plantaire pathologique, Rev. neurol. **28**:455, 1921.

178. Brain, W., and Curran, R.: The Grasp Reflex of the Foot, Brain **55**:347, 1932.

179. Grinker, R.: Neurology, Springfield, Ill., Charles C Thomas, Publisher, 1934, p. 243.

180. Walshe, F. M. R., and Ross, J.: The Clinical Picture of Minor Cord Lesions in Association with Injuries of the Cervical Spine, Brain **59**:277, 1936.

181. Gendron, A.: Étude clinique des tumeurs de la moelle et des méningés spinales, Thesis, Paris, no. 229, Paris, A. Maloine, 1913.

182. Footnote deleted by the author.

183. Babinski, J.: Réflexes tendineux et réflexes osseux, Bull. méd., Paris **26**:929, 1912.

184. André-Thomas, in discussion on Claude, H., and Velter, E.: Syringomyélie cervicale. Inversion du réflexe du radius, Rev. neurol. **20**:602, 1910.

185. Walshe, F. M. R.: Diseases of the Nervous System, Baltimore, Williams & Wilkins Company, 1940, p. 164.

tion is not due directly to the absence of certain other reflexes, but results from the concomitant involvement of the pyramidal tract. In no case is there any inversion or replacement. In other words, the absence of certain reflexes does not necessarily cause the exaggeration of others. Whether tapping of the radius will elicit, in addition to the flexion of the elbow, a flexion of the fingers depends on the position of the arm, the strength and direction of the blow, the general reflexive irritability and the presence of a pyramidal lesion above the seventh cervical segment—but by no means on the last condition only. Absence of one of the components of the multiple reflexes resulting from one tap should not be called inversion; rather, it is a dissociation, as Marie and Foix noted, due to the absence of one reflex and the presence of another. One does not speak of "inversion" of the patella reflex when, owing to a specifically localized lesion, there appears an adductor reflex but no patellar reflex when the patellar tendon is tapped.

BICEPS REFLEX

With the upper portion of the arm slightly abducted and the forearm bent, the biceps reflex is best tested by placing the distal phalanx of the thumb on the tendon of the biceps muscle and tapping the thumb. The biceps reflex may also be elicited from remote points, such as the lower third of the radius, the scapula and the acromion. The contraction of the biceps muscle constitutes a part of the acromial reflex of von Bechterew,[186] elicited by a tap on the acromion or the coracoid process. The biceps may occasionally contract when the pectoral reflex is elicited (see section on "Pectoral Reflex"). When the deep reflexes of the upper extremity are greatly increased, the biceps reflex may be elicited also on tapping of the clavicle, the manubrium sterni or even the olecranon or the clavicle on the opposite side. Myerson[173] devoted an article to the "contralateral periosteal reflexes of the arm"; these reflexes consisted of contraction of the biceps and pectoralis major muscles of the one arm when the middle of the opposite clavicle was tapped (previously observed by Westphal[175] and Strümpell[171]). It is true of the biceps reflex, as of any other deep muscle reflex, that if the threshold of a reflex is lowered the slightest direct or indirect concussion of the bones to which the muscle is attached, even from a remote point, elicits its reflex. A further method of elicitation of the biceps reflex will be discussed with the paradoxic triceps reflex.

186. von Bechterew, W.: Ueber den Acromionreflex, Neurol. Centralbl. **22**:194, 1903; footnote 144.

Galant[187] elicited the biceps reflex by exacting pressure on the middle of the muscle. In the same article in which he described the reflex, he himself named it the Galant biceps reflex, designating it as the twenty-first reflex of the upper extremity and claiming its "new discovery."

TRICEPS REFLEX

For better elicitation of this reflex it is necessary to bring the arm in such a position as to stretch the triceps muscle. With the patient lying prone, the arm is drawn across the chest, the forearm being slightly flexed. The tendon of the triceps muscle is tapped just above the olecranon. In order that the triceps reflex may be compared on the two sides, the patient may be asked to place his arms akimbo, his outspread hands resting at the waist above his hips (Zalkan and Frenkel[188]). From his position behind the patient, the examiner can easily compare the triceps reflex on the two sides, since the position of the arms is the same. Needless to say, when the triceps reflex is exaggerated it may be elicited from other, remote, points—for instance, the styloid process of the ulna or the dorsal surface of the distal part of the forearm, the forearm being bent at a right angle. Valobra and Bertolotti[189] succeeded in eliciting a contraction of the triceps muscle on the paralyzed side in a case of hemiplegia on tapping the spinal column in the lower cervical region. Bickel[70] reported bilateral contraction of the triceps muscle on tapping the lower cervical or the upper thoracic vertebrae in cases with increased reflexive irritability.

PARADOXIC TRICEPS REFLEX

The adjective "paradoxic" is popular for designation of reflexes which at first glance do not fit into any common pattern. Gordon,[190] for instance, called his reflex, a dorsiflexion of the big toe on deep pressure of the calf muscles, a "paradoxical reflex." There is nothing paradoxic about it. It is simply a modification of the technic for elicitation of the Babinski toe sign. In the neurologic literature, especially the French, there is much discussion of the "paradoxic triceps reflex." This phenomenon con-

187. Galant, J. S.: Ein neuer Bicepsreflex, Monatscher. f. Kinderh. **33**:110, 1926.

188. Zalkan and Frenkel: On the Method of Examination of the Tendon Reflexes of the Triceps, Vrach. delo **20**:54, 1938.

189. Valobra, J., and Bertolotti, M.: Ueber einige neue Knochenreflexe der unteren Gliedmaassen im gesunden und im pathologischen Zustande, Neurol. Centralbl. **24**:343, 1905.

190. Gordon, A.: A Study of Reflexes, J. Nerv. & Ment. Dis. **34**:430, 1907.

sists of a flexion, not an extension, of the forearm when the triceps tendon is tapped, as for elicitation of the triceps reflex.

When, in an attempt to elicit a deep muscle reflex, one obtains not the expected reflexive movement but some response of a quite different kind, there should first arise the question whether a stimulus capable of eliciting a muscle stretch reflex was applied or whether, with the technic employed, a superficial sensory stimulus only was given. It is easy to exclude any deep muscle reflex when the stimulus is superficial and is not likely to evoke stretching of a muscle or when the phenomena appear contralaterally or are elicited symmetrically in remote muscles that could not possibly undergo stretching (Neustadt and Sioli [191] and Mayer [192]). Under the last-named condition reflexes of defense or postural reflexes, and the like, but not muscle stretch reflexes, are evolved. Even when the reflexive movement appears in the same extremity, the complete action must be carefully analyzed for two factors: First, the stimulus applied must be such as to effect muscle stretching, and, second, the muscle actually contracting, and thus leading to the paradoxic reflex, must have been subject, directly or indirectly, to stretch by the blow of the reflex hammer. Herzog [193] tried to explain the paradoxic triceps reflex thus: As a result of a lesion of the arc of the triceps reflex, a stimulus of greater strength reaches other reflex tracts, i. e., those which lead to the flexors of the forearm, and thus a contraction occurs in these muscles. This explanation, aside from being too vague, does not account for the fact that it is precisely the antagonists of the triceps muscle, the flexors of the forearm, which contract in the paradoxic reflex, whereas no other muscles are involved. Kroll [194] asserted that the so-called paradoxic triceps reflex, as well as other paradoxic reflexes, is proof that there are proprioceptors not only in the muscles but in the neighboring structures which are capable of receiving stimuli leading to reflexive contraction of the muscles. Foerster,[195] too, explained the paradoxic reflex by the assumption that receptors in the tendons and the periosteum are stimulated and that this stimulation is relayed to the anterior horn cells of the corresponding muscles.

The paradoxic reflexes offer one of the best proofs that the receptors concerned with the so-called deep muscle reflexes lie in the muscles themselves, and there only, and that only brisk longitudinal stretching may evoke these reflexes.

If one recalls my previous analogy of the muscle and the bowstring, in which it was pointed out that stretching of the bowstring or of the muscle may be achieved by a force which acts either on the plane or on the convex side of the bow mechanism, one will easily understand that the so-called paradoxic reflex

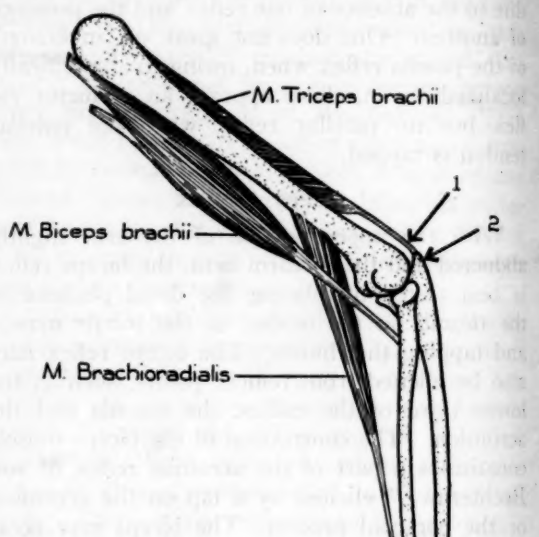

Fig. 5.—Schematic drawing illustrating the physiologic mechanism of the paradoxic triceps reflex. A blow of the reflex hammer in the direction of arrow *1* will effect essentially a stretching of the triceps muscle and elicit the triceps reflex. A blow in the direction of arrow *2* will essentially depress the elbow joint, stretch the flexors of the arm and elicit their reflex, leading to bending of the elbow—the paradoxic triceps reflex.

is an entirely normal phenomenon. In tapping the muscle on the convex side, one depresses the arc, and thus stretches the muscles strung over the concave side of the joint. This mechanical depression of the arc, eventually accompanied by the contraction of the agonist muscles, causes a stretching of the antagonist muscles and thus may, under certain circumstances, evoke their reflexive contraction (fig. 5). This stretching of the antagonists is so self evident and is clin-

191. Neustadt, R., and Sioli, F.: Ueber gekreuzte und diagonale Reflexe, Arch. f. Psychiat. **100**:141, 1933.

192. Mayer, C.: Beiderseitiger gekreuzter Grundgelenkreflex und andere gekreuzte Reflexe bei früh entstandener Gehirnerkrankung, Wien. klin. Wchnschr. **37**:515, 1924.

193. Herzog, F.: Sehnenreflexzeit und Muskeltonus, Reflexzeit des Babinskischen Zeichens, infolge von Lähmung peripherer Nerven gesteigerter Sehnenreflex, Neurol. Centralbl. **37**:225, 1918.

194. Kroll, M.: Die neuropathologischen Syndrome, Berlin, Julius Springer, 1929, p. 191.

195. Foerster, O.: Schlaffe und spastische Lähmung, in Bethe, A.; von Bergmann, G., and others: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1927, vol. 10, p. 893.

ically so apparent that I cannot follow Foerster and Altenburger,[196] who assumed that owing to the blow on the bone the points of insertion of the antagonists come closer together and the muscles are not stretched.

In his monograph on reflexes, Sternberg[8] stressed that on elicitation of the deep reflexes the antagonists nearly always contract with the muscle the tendon of which is tapped. He noticed that often in normal persons the biceps contracted with the triceps on elicitation of the triceps reflex. To this contraction of the antagonists he attributed the fact that a powerful, energetic contraction of the quadriceps muscle often provokes only a slight movement of the lower portion of the leg. Physiologists noticed long ago that on elicitation of the patellar reflex the flexors of the knee may contract too (Lombard,[197] Pfahl,[198] Golla and Hettwer[199]). The same is true of the achilles reflex.

It is not an unusual clinical experience to find that these paradoxic contractions can be more easily seen when the patellar or the achilles reflex is absent; then the tapping of the patellar tendon evokes contraction of the biceps femoris muscle, and the tapping of the achilles tendon evokes contraction of the tibialis anticus and the extensor hallucis longus muscle. (Foerster[200]). These reflexes of the antagonist muscles can by proper technic be elicited in normal persons—especially in neurotic subjects—with exaggerated reflexes (Barré and Draganesco,[201] Egas Moniz,[202] and Stern[203]). But they are usually not pronounced because the contraction of the agonist muscles—reflexes of which are primarily elicited—prevents the contraction of the antagonist muscles. But the reflexive contraction of the antagonists becomes manifest when the relation between the muscle tonus of the agonist and that of the antagonist changes in favor of the antagonist. This can occur when the tonus and the reflex of the agonist muscle are decreased or when the tonus, and necessarily the reflex, of the antagonist muscle are increased. Everything depends on which of the two competitive muscles, the flexors or the extensors, shows greater responsiveness to a stretch stimulus.

The so-called paradoxic triceps reflex is found first in all cases in which the arc of the triceps reflex is damaged and the triceps reflex is diminished or absent. Here the blow on the convex side of the aforementioned imaginary bow, dealt to elicit the triceps reflex, can effect a stretching of the flexors, unopposed by reflexive action of the triceps muscle, which would contract normally. Thus, the paradoxic triceps reflex is present in cases of transverse lesions of the spinal cord at the level of the seventh and eighth cervical segments; the triceps reflex is absent, and an attempt to elicit this reflex by tapping the tendon of the muscle or the olecranon results in flexion of the forearm by indirect stretching of the muscles, the reflex arc of which lies higher, in the fifth and sixth cervical segments. This, by the way, constitutes a reliable sign of a transverse lesion at this level.

I observed this paradoxic reflex distinctly in a case of tumor of the seventh cervical segment of the spinal cord, confirmed by operation, in which the triceps reflex was absent and the brachioradial reflex was exaggerated, appearing on the slightest tapping on any part of the convex side of the elbow, depressing it or bringing about its concussion. Careful observation admitted no doubt as to the mechanism of this phenomenon. This paradoxic triceps reflex is seen with any lesion which involves the seventh and eighth cervical segments but leaves the fifth and sixth segments intact, as may occur in poliomyelitis, hematomyelia, syringomyelia, myelitis, meningitis, radiculitis and neuritis (Lorenz,[204] Mankowsky and Beder[205] and Sternberg[8]). It is interesting to note that when the lesion spreads upward and involves the fifth and sixth cervical segments, the paradoxic reflex disappears. Not uncommonly, the paradoxic triceps reflex is seen in cases of tabes, in which the triceps reflex may often be lost early while the flexor reflexes of the arm are preserved. This is exactly the physiologic condition which makes the paradoxic reflex appear distinctly. It is therefore understandable that this reflex has often been described

196. Foerster, O., and Altenburger, H.: Zur Physiologie and Pathophysiologie der Sehnen- und Knochenphänomene, Ztschr. f. d. ges. Neurol. u. Psychiat. **146**: 641, 1933.

197. Lombard, W. P.: On the Nature of the Knee-Jerk, J. Physiol. **10**:122, 1889.

198. Pfahl, J.: Beiträge zur Physiologie der Sehnenreflexe, Ztschr. f. d. ges. Neurol. u. Psychiat. **1**:350, 1910.

199. Golla, F., and Hettwer, J.: Influence of Various Conditions on Time Relation of Tendon Reflexes in the Human Subject, Proc. Roy. Soc., London, s.B, 1922, p. 92.

200. Foerster,[131] p. 266.

201. Barré and Draganesco: Inversion des deux réflexes tricipitaux, fracture du radius d'un côté, Rev. neurol. **2**:467, 1926.

202. Egas Moniz: Réflexes du coude chez les hémiplégiques, Rev. neurol. **2**:759, 1912.

203. Stern, F.: Ueber gekreuzte und paradoxe Reflexe, Zentralbl. f. d. ges. Neurol. u. Psychiat. **33**:398, 1923.

204. Lorenz, H. E.: Beitrag zur Kenntnis der paradoxen Reflexe, Deutsche Ztschr. f. Nervenh. **54**:259, 1915.

205. Mankowsky, B. N., and Beder, W. L.: Ueber paradoxale Reflexe, Monatschr. f. Psychiat. u. Neurol. **86**:166, 1933.

in cases of tabes (Foerster,[206] Souques,[207] Lian,[208] Lorenz,[204] Benedek and de Thurzó [209] and Mankowsky and Beder [205]).

The paradoxic triceps reflex appears distinctly under all those pathologic conditions which change the balance of the muscle tonus of the extensors and flexors of the forearm in favor of the flexors. Thus, as has just been shown, this reflex appears when the triceps reflex is lost and the flexor reflex of the forearm either remains normal or is exaggerated. The relation of the flexor and the extensor tonus may be disturbed in that the tonus muscle of the triceps may remain normal or be exaggerated, but the exaggeration of the tonus may be greater on the part of the flexors. This condition is found in every case of spastic hemiplegia, in which, as is common knowledge, the flexors are more spastic than the extensors and the flexor reflexes of the arm are more exaggerated than the triceps reflex. Therefore it is not surprising that the paradoxic triceps reflex has often been noted in cases of hemiplegia (Babinski,[210] Sablé,[211] Foerster [206] and Böhme [212]). Egas Moniz [202] found it in 80 per cent of his cases of hemiplegia. In cases of spastic paralysis I have observed on repeated examinations that it was possible by a single tap to elicit either the triceps reflex alone or the flexor reflex of the forearm alone or both. Here all depended on the point at which the tap was applied, its direction and intensity, and particularly the position of the forearm, i. e., the angle of flexion. Similar observations have left no doubt in my mind that it is always the indirect longitudinal stretching of the flexor muscles which produces the so-called paradoxic reflex. Babinski [210] was certainly in error when, in explaining the paradoxic triceps reflex, he stated: "The flexion is due, in effect, to an excitation not of the tendon but of the underlying bone; this is a bone reflex."

206. Foerster, O.: Zur Symptomatologie der Tabes dorsalis, Monatschr. f. Psychiat. u. Neurol. **8**:1, 1900.

207. Souques, M. A.: Inversion du réflexe tendineux du triceps brachial dans l'hémiplégie associée au tabes, Rev. neurol. **21**:510, 1911.

208. Lian, C.: Inversion du réflexe tricipital chez une tabétique, Rev. neurol. **23**:436, 1912.

209. Benedek, L., and de Thurzó, E.: Du réflexe paradoxal des triceps et de sa localisation segmentaire, Rev. neurol. **2**:463, 1927.

210. Babinski, M. J., in discussion on Souques, M. A.: Inversion du réflexe tendineux du triceps brachial dans l'hémiplégie associée au tabes, Rev. neurol. **21**:511, 1911.

211. Sablé, M. J.: Inversion du réflexe tricipital au cours d'une hémiplégie organique, Rev. neurol. **21**:640, 1911.

212. Böhme, A.: Das Verhalten der Antagonisten bei spinalen Reflexen und die Reflexumkehr, Deutsche Ztschr. f. Nervenh. **56**:256. 1917.

Nor can one agree with Foerster [213] or Wachholder and Altenburger,[214] who assumed that the antagonists contract because the stimulus is not confined to the center of the corresponding muscle but flashes across the spinal cord to the centers of other muscles. There is no need to assume such central transmission. The tap on the bone to elicit the reflex of the agonists, the depression of the joint and the reflexive contraction of the agonists, all these constitute a stimulus powerful enough to bring about, purely peripherally, concussion and stretching of the antagonists. Minkowski [215] explained, rather vaguely, the paradoxic reflexes as idiomuscular phenomena appearing because of a weakened influence of the spinal cord on the muscles. But paradoxic phenomena are not idiomuscular in their origin, nor do they appear only when there is a weakening of the influence of the spinal cord on the muscles.

From what has been said, it is easily understood that the most favorable condition for the appearance of the paradoxic triceps reflex is that in which tabes is combined with spastic hemiplegia. No wonder that the paradoxic triceps reflex was first described by Souques,[207] in 1911, in a case of hemiplegia associated with tabes.

Dejerine,[216] in his classic textbook, as well as Pitres and Testut,[217] mentioned an "olecranon reflex," which consisted of flexion of the forearm when the posterior aspect of the olecranon was tapped. This technic constitutes only one of the methods of elicitation of the flexor reflex of the forearm and is practically nothing but the "paradoxic triceps reflex." It is not an osteoperiosteal reflex, in contradistinction to the tendon reflexes, as the French authors assumed, but is simply a deep muscle reflex of the flexors of the forearm, elicited by special technic. The term "olecranon reflex" is not correct, since the same reflex can be elicited, for instance, from the dorsal aspect of the upper part of the ulna. Schulz (cited by Sternberg [8]) described three reflexes: the olecranon, the epicondylus internus humeri and the condylus externus humeri reflex, all leading to contraction of the biceps and triceps muscles.

213. Foerster, O., in Lewandowsky, M.: Handbuch der Neurologie, Berlin, Julius Springer, 1929, supp., pt. 2, p. 1221.

214. Wachholder, K., and Altenburger, H.: Ueber die Wechselbeziehungen zwischen den Sehnenreflexen und der antagonistischen Innervation unserer Muskeln, Arch. f. d. ges. Physiol. **203**:620, 1924.

215. Minkowski, M.: Neurobiologische Studien am menschlichen Foetus, in Abderhalden, E.: Handbuch der biologischen Arbeitsmethoden, Berlin, Urban & Schwarzenberg, 1938, vol. 5, pt. 5 B.

216. Dejerine,[156] p. 949.

217. Pitres and Testut,[140] p. 683.

It is evident therefore that there is nothing paradoxic about the so-called paradoxic triceps reflex. The term "paradoxic" is itself misleading. This is a reflex which can be elicited in normal persons but is usually seen in persons with pathologic conditions. It represents only a modification of the usual technic for elicitation of the flexor reflex of the forearm. With reference to the analogy between the reflex mechanism of the elbow and a bowstring, the string representing the flexors of the forearm, I may point out that it is just as paradoxic to elicit the flexor reflex by tapping the elbow as it is to produce stretching of the bowstring by tapping the top of the bow. The paradoxic triceps reflex is just as "paradoxic" as von Bechterew's carpometacarpal reflex, discussed earlier. This normal reflex is elicited on the same principle as the paradoxic triceps reflex: Tapping the dorsal aspect of the hand evokes flexion of the fingers.

Many other reflexes can be elicited, not only through direct, but through indirect, stretching of the muscles, by depressing the joints over which they pass. What has been said of the paradoxic triceps reflex applies unequivocably to other reflexes—for instance, to the patellar and to the achilles reflex. The knee flexors may participate normally on elicitation of the patellar reflex. This is true particularly when the contraction of the flexors is not hampered by the contraction of the extensors, i. e., when the patellar reflex is lost; then an attempt to elicit the patellar reflex may evoke a bending of the knee. This has often been observed, for instance, with tabes, poliomyelitis, nerve resection and all other lesions affecting the arc of the patellar reflex, as well as occasionally with lesions of the pyramidal tract, as in cases of multiple sclerosis (Dejerine and Jumentié,[218] Sittig,[219] Lorenz,[204] Guber-Gritz,[220] Yakovlev [221] [in the case of postencephalitic parkinsonism] and Mankowsky and Beder [205]). Flexion of the knee on elicitation of the patellar reflex was observed by Berger,[222] in 1879, and by Benedikt,[223] in 1889. The latter used for the first time the term "paradoxic." Yet, in 1911, Dejerine and Jumentié,[218] in reporting a case of inversion of the patellar reflex accompanying tabes, stated that they could find nothing similar previously described in the literature. In cases of disturbances of the crural nerve, Foerster and Altenburger [196] noted repeatedly a paradoxic patellar reflex, namely, contraction of the biceps, semitendinosus and semimembranosus muscles on tapping the patellar tendon. Later, they verified this observation by study of the behavior of antagonist muscles on other joints. Many observers of this paradoxic patellar reflex have stressed the fact that a great deal depends on the position of the extremity and the exact point and direction of the blow. This is understandable, since in order to elicit a flexor reflex by tapping the convex side of the knee one must direct the blow as nearly as possible at a right angle to the flexors. In this way, stretching of the flexors is best effected. The paradoxic triceps reflex is much more often observed than the paradoxic patellar reflex. The reason for this is simple—the knee joint is much more bulky and therefore much more difficult to move by percussion with a reflex hammer. It is much easier by a single tap to depress the elbow joint than the kee joint. It is also easier to bring about a percussion of the bones of the arm than those of the leg. If the dorsiflexors and plantar flexors of the foot are so affected that the balance of their tonus is changed in favor of the dorsiflexors, one might, on tapping the achilles tendon, effect a dorsiflexion of the foot more easily than a plantar flexion. This, again, is a paradoxic phenomenon, a paradoxic achilles reflex, as has occasionally been described (Lorenz,[204] Russetzki,[224] Debray,[225] Mankowsky and Beder,[205] Kastein,[226] Foerster [200]). Böhme [227] stressed the fact that this can be seen also in normal persons.

218. Dejerine, J., and Jumentié, J.: Inversion des réflexes patellaires dans un cas de tabes, Rev. neurol. **19**:725, 1911.

219. Sittig, O.: Wiederkehr des Patellarreflexes in einem Falle von hoher Rückenmarksdurchtrennung und die lokalisatorische Bedeutung des sogannanten paradoxen Kniephänomens, Monatschr. f. Psychiat. u. Neurol. **38**:257, 1915.

220. Guber-Gritz, D, S.: On Inversion of Reflexes, Zhur. nevropat. i psikiat. **22**:45, 1929.

221. Yakovlev, P. I.: Paradoxical Forms of the Tendon Reflexes and Their Clinical and Physiological Significance, Bull. Massachusetts Dept. Ment. Dis. **16**: 11, 1932.

222. Berger: Ueber Sehnenreflexe, Centralbl. f. Nervenh. u. Psychiat. **2**:73, 1879.

223. Benedikt, M.: Einige qualitative Varietäten des Kniephänomens, Neurol. Centralbl. **8**:489, 1889.

224. Russetzki, J.: Le syndrome pyramidal, Acta med. Scandinav. **73**:260, 1930.

225. Debray: Réflexe achilléen paradoxal, J. de neurol. et de psychiat. **6**:276, 1901.

226. Kastein, G. W.: Klinische Untersuchungen über die Receptoren der Sehnen- und Periostreflexe und über die Art der "paradoxen" Reflexe und des kontralateralen Adductorenreflexes, Nervenarzt **9**:105, 1936.

227. Böhme, A.: Vergleichende Untersuchungen über die reflektorischen Leistungen des menschlichen und des tierischen Rückenmarks, Deutsches Arch. f. klin. Med. **121**:129, 1916.

PECTORAL REFLEX

This is a muscle stretch reflex of the pectoral muscle and can be elicited by direct longitudinal stretching of the muscle, both at its origin and at its insertion, or by indirect stretching. The attachment of this muscle to the highly elastic thorax makes it understandable that the pectoral reflex can easily be elicited contralaterally, as well as bilaterally. Von Bechterew [228] described this reflex, and Noto,[229] in 1930, named it the "costopectoral reflex." His observations were elaborated on by Pelliccioli.[172] The reflex is to be elicited by tapping the seventh rib between the anterior and the medial axillary line; the arm should be slightly abducted and supinated, or, preferably, it should be placed above the head. All this aims at putting the pectoral muscle in a position of stretch before elicitation of the reflex. This reflex can also be elicited by another method: The arm of the patient is held in abduction, and the finger of the examiner is placed over the tendon of the pectoral muscle at the point of its origin on the humerus; the finger is pressed against the humerus and then tapped. The resulting reflexive action of the muscle brings about an adduction and slight internal rotation of the arm. The anterior portion of the deltoid muscle often contracts, too, as does the biceps muscle occasionally. This reflex is normal, though often weak and not easily elicited. In rare instances it is absent, as it was in 14 of the 316 cases reported by Pelliccioli.[172] In cases of hemiplegia it behaves like other deep muscle reflexes. In cases of functional or organic hyperirritability this reflex can be elicited bilaterally by tapping the sternum or by tapping the abdominal wall, as for elicitation of the abdominal muscle reflex. Here the thoracic cage to which the pectoral muscle is attached is indirectly jarred by the sudden brisk stretching and contraction of the abdominal muscles. The sternobrachial reflex described by Smirnoff,[230] which is elicited by tapping the sternum, has as its component the adduction of the arm. The pectoral reflex is a part of this reflex. The sternobrachial reflex is not in itself pathologic, as Smirnoff [230] asserted it to be.

In cases of spastic tetraplegia, tapping of the sternum or of the abdominal muscles evokes a concussion of the muscles attached to the thorax and of the muscles of the arm, since the concussion is easily transmitted to the scapula and the humerus. A complicated reflex action involving the muscles of the thorax, the shoulder girdle and the arms results. Contraction may even reach the flexors of the fingers. Chodzko, in 1909 and 1936,[174] reported a "sternal sign" which consisted of contraction of the muscles of the upper extremities when the sternum was tapped. The pectoral muscle reflex is a prominent part of Chodzko's sternal sign. It is identical with Smirnoff's sternobrachial reflex. Strümpell [171] and Myerson [173] observed that the contralateral pectoralis muscle contracted when the clavicle was tapped. Pickett [231] noted strong contraction of the pectoralis major muscle when the inner edge of the scapula was tapped. Even tapping of the wrist joint may evoke the pectoral reflex.

SCAPULOHUMERAL REFLEX

On tapping the vertebral border of the scapula, particularly its inferior part, one can evoke reflexive contractions of different degrees of strength in a multitude of muscles of the shoulder girdle and the arm. These reflexive contractions vary in range and intensity according to the strength, the direction and the exact point of application of the tap and the position of the trunk, scapula and humerus. The position of the scapula here is an especially important factor. The muscles exposed to the greatest stretching give the greatest response. It is impossible to name this reflex according to the muscles in action; so it is advisable to keep the name given it by von Bechterew,[232] in 1900—the scapulohumeral reflex. Steinhausen [233] called it the scapuloperiosteal reflex. The infraspinatus reflex of Steiner [234] is identical with this reflex. The following muscles may be called into action by the tap on the border of the scapula: the deltoid (especially the posterior portion), the pectoralis major, the infraspinatus, the teres minor and, eventually, the flexors of the forearm—even those of the wrist and of the fingers. Some authors say that next to the outward rotation of the forearm is adduction of the arm; others claim abduction. According to von Bechterew,[232a] the reflex consists of an adduction of

228. von Bechterew, V.: On the Subscapular and Pectoral Reflex, Obozr. psikhiat., nevrol. **7**:899, 1902.

229. Noto, G. G.: Un nuovo riflesso: il riflesso costo-pettorale, Boll. Soc. ital. di biol. sper. **5**:508, 1930; Il riflesso costo-pettorale, Riv. di pat. nerv. **40**:477, 1932.

230. Smirnoff, B.: Le réflexe sterno-brachial, Rev. neurol. **2**:659, 1934.

231. Pickett, W., in discussion on Mills and McConnell: Brachial and Pectoral Reflexes, J. Nerv. & Ment. Dis. **29**:429, 1902.

232. von Bechterew, W.: (*a*) Ueber den Scapulo-Humeralreflex, Neurol. Centralbl. **19**:208, 1900; (*b*) Ueber die Bedeutung des Scapulo-Humeralreflexes, ibid. **19**:1042, 1900.

233. Steinhausen: Ueber den Scapula-Periostreflex, Neurol. Centralbl. **20**:507, 1901.

234. Steiner: Der Infraspinatusreflex, Neurol. Centralbl. **21**:840, 1902.

the arm, but abduction takes place not infrequently. The latter, I think, is more common—especially on stronger tapping. Pickett [235] stated:

> The most frequent combination of motions, among all cases, was abduction of the upper arm with flexion of the forearm.

Cacciapuoti,[236] in a recent article, stressed, too, the resulting abduction of the arm. In any event, it is hardly possible to say that the reflex is pathologic and indicative of a pyramidal lesion if there is abduction of the forearm or any slight modification in the complex movement that follows the tap on the scapular border, as some authors have claimed (Frey,[237] Darkshevitch,[238] Grossmann,[239] Steinhausen [233] and Cacciapuoti [236a]). The participants in this reflex and the resulting movements are extremely varied and show no constancy. These movements show no definite qualitative changes with lesions of the pyramidal tract. With such lesions "the scapulohumeral reflex may spread far beyond its customary range," as Wilson [240] put it. The scapulohumeral reflex has been observed to be constant by some authors (Kollarits,[241] Steinhausen,[233] Kornilow [81] and Tiefensee [242]), less so by others (Haenel [243] and Pickett [235]). Its significance was questioned by Haenel [243] and Kornilow.[81] Its value is indeed not great, since it is difficult to analyze the complex movements which follow the tap on the scapular border and the reaction may be weak.

It is worth while, however, to emphasize the clinical importance of the unilateral absence of this reflex, since this may be the chief indicator of a lesion of the fifth cervical segment, and not, as Pitres and Testut [217] stated, of the fourth cervical segment. I have seen cases in which this absence was pronounced, conspicuous and diagnostically of great help.

235. Pickett, W.: The Scapulo-Humeral Reflex of von Bechterew, J. Nerv. & Ment. Dis. **28**:273, 1901.

236. Cacciapuoti, G. B.: (*a*) Sul riflesso marginoscapolare in condizioni normali e patologiche, Riv. di pat. nerv. **43**:162, 1934; (*b*) Sul riflesso marginoscapolare in condizioni normali e patologiche, Osp. psichiat. **1**:55, 1933.

237. Frey, E.: Ueber den Infraspinatusreflex, Ztschr. f. d. ges. Neurol. u. Psychiat. **1**:20, 1910.

238. Darkshevitch, cited by Russetzki.[224]

239. Grossmann, J.: Les nouveaux réflexes pathologiques, Rev. neurol. **1**:451, 1929.

240. Wilson,[189] p. 1013.

241. Kollarits, J.: Das Verhalten einiger Reflexe bei Gesunden und bei Tabes, Deutsche Ztschr. f. Nervenh. **23**:89, 1903.

242. Tiefensee, K.: Die Reflexe an den oberen Extremitäten, Arch. f. Psychiat. **74**:52, 1925.

243. Haenel, H.: Ueber den Scapulo-Humeralreflex, Neurol. Centralbl. **19**:399, 1900.

ABDOMINAL SKIN REFLEX

The abdominal skin reflex, which is generally called the "abdominal reflex," was first described by Rosenbach [244] in 1876. The latest, and most comprehensive, review on this reflex is that of van Rijnberk.[245] It is the classic representative of a skin reflex. The term "skin" reflex seems most appropriate since it is the skin that is the point of origin of this reflex. On mechanical irritation of the skin of the abdomen a visible contraction of the muscles takes place, resulting in lateral deviation of the umbilicus and the linea alba. In doubtful cases one should ascertain the contraction of the abdominal wall, not only by inspection but by palpation. This reflex varies in its intensity with different persons much more than do the deep muscle reflexes. Physiologically, by stiffening the abdominal muscles, it serves the useful purpose of protecting the viscera against external impact of any kind and of maintaining the endangered erect posture. In man, as a biped, the viscera are especially exposed to damage from without. The fact that the quadrupeds, even the anthropoid apes according to some investigators, have no abdominal skin reflexes may speak for a possible connection of these reflexes with the erect posture of man.

To elicit the abdominal skin reflex, long longitudinal or transverse strokes are applied to the skin of the abdomen, a summation of stimuli thus being produced. The essence of the stimulus lies in the quickness of the stroke; one can hardly elicit the abdominal skin reflex by slowly performed strokes. If the abdominal skin reflex is weak or absent, quicker, brisker strokes should be tried. The reflex may then become stronger, or if previously absent, it may then appear. The reflexogenous zone for this reflex reaches beyond the skin of the abdomen and may extend upward to the skin of the chest or downward to the inner upper part of the thigh, or even farther. Even in this case there is no need to speak of the thoracoabdominal reflex, as Barkman [246] did, or of the femoroabdominal reflex, as Dujardin [247] did. The thoracoabdominal reflex, i. e., contraction of the abdominal wall on stimulation of the skin above the costal margin, where no more abdominal

244. Rosenbach, O.: Ein Beitrag zur Symptomatologie cerebraler Hemiplegien, Arch. f. Psychiat. **6**:845, 1876.

245. van Rijnberk, G.: Les réflexes de la paroi abdominale, Arch. néerl. de physiol. **22**:457, 1937.

246. Barkman, Å.: Sur les réflexes thoraco-abdominaux normaux et leur localisation médullaire, Acta med. Scandinav. **58**:364, 1923.

247. Dujardin, B.: Le réflexe fémoro-abdominal au cours du tabes, J. de neurol. et de psychiat. **26**:295, 1926.

muscle fibers are present, had been observed before by Stern-Piper [248] and by Petrén,[249] who did not resort to the use of new names. The femoroabdominal reflex of Dujardin is nothing but the hypogastric reflex described by von Bechterew [250] in 1901 (discussed by Ferrio [251] and Kornilow [81]), but observed before him by Geigel [252] and Dinkler,[253] in 1892; by Strümpell,[254] in 1899, and van Gehuchten,[255] in 1900. Crocq,[256] in 1901, called this response the "inguinoabdominal" reflex. In 1918 it was rediscovered by Rodella.[257] After all this, it is surprising to learn that Galant,[258] in 1928, described a "cremasteric-abdominal reflex" to which he wished to draw attention, since he had not found it mentioned before. This "new" reflex consisted of contraction of the abdominal wall on elicitation of the cremaster reflex by stroking the skin on the inner side of the thigh!

There would be no end to names of reflexes if each one were to be designated as new when it could be elicited from some point more distant than the usual area. This is particularly true of the abdominal skin reflex, since its reflexogenous area, like that of another cutaneous reflex, the cremasteric, may be very extensive. In cases of tabes, particularly during gastric crises, in which the abdominal skin reflexes are usually exaggerated, these responses may be elicited from the anterior surface of the thigh, from the lower portion of the leg, from the upper part of the thorax and even from the back of the trunk; they may be elicited, also, by pressure on the testicles. What an opportunity to describe new reflexes as plantoabdominal, dorsoabdominal and testicoabdominal!

248. Stern-Piper: Ueber Bauchdeckenreflexe, München. med. Wchnschr. **68**:1421, 1921.

249. Petrén: Bemerkung zum nosographischen Verhalten der Bauchreflexe, Deutsche Ztschr. f. Nervenh. **70**:91, 1921.

250. von Bechterew: Ueber den hypogastrischen Reflex, Neurol. Centralbl. **20**:647, 1901.

251. Ferrio, L.: Sul riflesso ipogastrico di Bechterew, Riv. crit. di clin. med. **3**:69, 1902.

252. Geigel, R.: Die klinische Prüfung der Hautreflexe, Deutsche med. Wchnschr. **18**:166, 1892.

253. Dinkler, M.: Ueber die Localisation und das klinische Verhalten der Bauchreflexe, Deutsche Ztschr. f. Nervenh. **2**:325, 1892.

254. Strümpell, A.: Ueber das Verhalten der Haut- und Sehnenreflexe, Neurol. Centralbl. **18**:617, 1899.

255. van Gehuchten, A.: Réflexes cutanés et réflexes tendineux, Nevraxe **1**:247, 1900.

256. Crocq, J.: Dissociation et antagonisme des réflexes tendineux et cutanés, J. de neurol. **6**:45, 1901.

257. Rodella, A.: Ueber konkomitierende Reflexe, Cor.-Bl. f. schweiz. Aerzte **48**:1102, 1918.

258. Galant, J.: Ueber den Kremaster-Bauchreflex, Fortschr. d. Med. **46**:588, 1928.

For stimulation of the skin of the abdomen, the following instruments may be used: a dull pin, a pencil, a match, a key, a brush, the handle of the percussion hammer, a strip of paper or the finger nail. Some examiners recommend other stimuli, such as pinching, tickling, slight percussion and application of heat or cold, or of faradic current in the form of a small roller.

The application of sharp instruments for elicitation of the abdominal skin reflex has its disadvantages. First, it hurts the patient, sometimes producing painful, visible scratches on the skin. The second disadvantage was described by Monrad-Krohn [259] as follows:

> . . . When the abdominal wall is fat and flabby, the stroke often sets up an undulating movement in the panniculus adiposus, which makes it difficult to decide whether there is any muscular reaction at all.

This "wave" in the fat abdominal wall may also conceal or simulate a possible deviation of the umbilicus. To avoid both these disadvantages, "drying of the surface and dusting with talcum powder" has been suggested by Skinner.[260] But these disadvantges may be obviated more readily by use of the pinwheel described in a former communication.[261] In the examination for abdominal skin reflexes, the pinwheel is quickly rolled over the abdomen under light pressure. Many years' experience has taught me that this method is more effective than any other. It has often been observed that an abdominal skin reflex which could not be elicited by any other method could be clearly seen when the pinwheel was used. An abdominal skin reflex should not be declared lost until such an examination has been made. Bing [262] stated: "When weak stimuli fail, the abdominal reflexes may come to light on the use of the pinwheel recommended by Wartenberg."

Complete relaxation or too great tension of the abdominal muscles may abolish the abdominal skin reflex. A medium tension is most favorable. If the abdominal walls are too relaxed, it may be advisable to have the patient contract the muscles slightly or to examine him in a sitting position, in which the sinking of the viscera brings about some tension of the abdominal wall. I saw Cassirer examine for the abdominal skin reflex in the following manner: He placed his hand below the umbilicus at a right angle to the abdomen, exerting slight

259. Monrad-Krohn,[77] p. 141.

260. Skinner, E. F.: The Superficial Abdominal Reflexes, J. Ment. Sc. **82**:394, 1936.

261. Wartenberg, R.: A Pinwheel for Neurologic Examination, J. A. M. A. **109**:1294 (Oct. 6) 1937.

262. Bing,[117] p. 200.

pressure toward the symphysis and thus stretching the abdominal muscles; at the same time he lightly stroked the skin of the abdomen. This method was mentioned by Blum.[263]

With irritative lesions of the cortex the contralateral abdominal skin reflex may be exaggerated (Redlich[264]), but the greatest diagnostic significance of the abdominal skin reflex lies in the fact that it is abolished in the presence of a lesion of the pyradimal tract; this constitutes a valuable pyramidal sign. Yanowitz[265] observed that lesions of the frontal and parietal lobes are especially likely to abolish the abdominal skin reflex. Strümpell[266] pointed out the early abolition of the abdominal skin reflex in cases of multiple sclerosis, an abolition which resists remissions.

According to Brain,[109] in a lesion of the pyramidal tract the abdominal (skin) reflexes of the lowest segments suffer most. But Nielsen[267] emphasized that loss of the upper abdominal (skin) reflexes with preservation of the lower ones is a characteristic symptom of multiple sclerosis. Only further observations will bring clarity on this important point.

When one is evaluating the absence of the abdominal skin reflex as a pyramidal sign, one must keep in mind that it may be absent under various conditions. It may be absent, first, with any local disturbance of the corresponding segments or of their afferent and efferent tracts. The abdominal skin reflex may be normally absent, also, in the first six to twelve months of life and in very old people. It may also be absent in people with fat, flabby, edematous abdominal muscles or after repeated pregnancies. Diffuse and localized, predominantly acute disease of the abdominal cavity, including the pelvis, and pathologic conditions causing pain near the abdominal wall, may all bring about loss of the abdominal skin reflex or its easy exhaustibility (Bott,[268] Goldflam,[269] Jamin,[270] Müller and Seidelmann,[271] Williams,[272] Olivecrona[273] and van Rijnberk[245]). This exhaustibility should be especially kept in mind, since it is a characteristic of this reflex even under normal conditions. Repeated elicitation causes the reflex to be less conspicuous, or even absent. It is, therefore, the result of the first examination that should be decisive.

In evaluation of the behavior of the abdominal skin reflex in the presence of a pyramidal lesion, it must be remembered that even when there is definite involvement of the pyramidal tracts, the abdominal skin reflex may still be present, or may return soon. This is true especially with such pathologic conditions as amyotrophic lateral sclerosis, spastic paralysis, infantile hemiplegia and congenital spastic diplegia (Little). It should further be kept in mind that the abdominal skin reflex may return before the lesion is healed, that its strength is not proportional to the severity of the pyramidal lesion and that in cases of multiple sclerosis it may be lost very early, when all other signs of pyramidal involvement are lacking.

ABDOMINAL MUSCLE REFLEX

Not only do the abdominal muscles have a reflex which originates outside themselves, in the skin, but they have, as do all other striated muscles, a reflex of their own, so to speak—an intrinsic reflex, a muscle-muscle reflex, a muscle stretch reflex. This is the abdominal muscle reflex. In other words, the abdominal muscles have two kinds of reflexes—dermatogenic and musculogenic. In the latter type, now under discussion, the abdominal muscles contract on direct or indirect stretching of the abdominal muscles themselves. On this sudden stretching, they contract in exactly the same way as does the quadriceps femoris muscle when the patellar tendon is tapped. Physiologically, this reflex is apparently closely related to the mechanism of erect posture.

263. Blum, D.: Zum Nachweis der Bauchreflexe, Neurol. Centralbl. **39**:358, 1920.

264. Redlich, E.: Ueber Steigerung der Hautreflexe an der paretischen Seite bei organischen Hemiparesen, Neurol. Centralbl. **24**:395, 1905.

265. Yanowitz, B.: Klinische Untersuchungen über die Bauchdecken- und Cremasterreflexe, Dissert., Zurich, Stampfenbach, 1937.

266. Strümpell, A.: Zur Pathologie der multiplen Sklerose, Neurol. Centralbl. **15**:961, 1896. This is the article of Strümpell in which he first mentioned the absence of "abdominal reflexes" in multiple sclerosis, and to which G. W. Hall (Neurologic Signs and Their Discoverers, J. A. M. A. **95**:703 [Sept. 6] 1930) refers.

267. Nielsen, J. M.: A Textbook of Clinical Neurology, New York, Paul B. Hoeber, Inc., 1941, p. 82.

268. Bott, G.: Einseitiges oder segmentäres Abschwächen des Bauchdeckenreflexes als Krankheitszeichen, Wien. klin. Wchnschr. **50**:1362, 1937.

269. Goldflam, S.: Sur la sémiologie des réflexes abdominaux, Rev. de méd., Paris **40**:547, 1923.

270. Jamin, F.: Ueber das Verhalten der Bauchdeckenreflexe bei Erkrankungen der Abdominalorgane, Monatschr. f. Psychiat. u. Neurol. **16**:289, 1904.

271. Müller, E., and Seidelmann, W.: Zur Physiologie und Pathologie der Bauchdeckenreflexe, München. med. Wchnschr. **52**:1323, 1905.

272. Williams, G.: The Loss of the Abdominal Reflexes in Affections of the Abdomen, Brit. J. Surg. **17**:320, 1919-1920.

273. Olivecrona, H.: Des réflexes abdominaux et de leur valeur diagnostique dans les affections aiguës de l'abdomen, Acta chir. Scandinav. **62**:185, 1927.

Owing to the vast expanse of the abdominal muscles, to their extensive line of origin and insertion—their long coast line, so to speak—which is far greater than that of any other muscle of the body, there are innumerable points from which they can be stretched and the reflex elicited. There are three main areas from which the muscles of the abdominal wall may be stretched: the costal margin, the line of insertion on the pelvis and the surface of the muscles themselves. The costal margin is best tapped at the nipple line. The stretching of the abdominal muscles produced by tapping the very elastic costal margin is so evident, so easily conceivable and perceivable, that it is hardly necessary to criticize the statement of Kroll [274] that it is impossible to bring about a stretching or concussion of the abdominal muscles by tapping the ribs. At their point of insertion on the pelvis, the abdominal muscles may best be stretched by percussion of the crista iliaca, the spina iliaca anterior superior, the ramus superior ossium pubis, the symphysis ossium pubis or the ligamentum inguinale (Poupart's ligament). The muscles themselves may be subjected to sudden stretching by a tap on the flat of the hand or on a ruler placed over them. The abdominal muscle reflex is seen distinctly when, with the patient lying on his back, a ruler is placed with slight pressure flat and horizontally across the muscle and briskly tapped. The "jumping" of the depressed ruler, due to sudden contraction of the muscle in cases in which the abdominal muscle reflexes are increased, is sometimes striking. If the muscles are struck suddenly and with force, their deep reflexes appear as powerful muscular contractions. Dumpert and Flick [166] described the following athletic stunt: If a man jumps from the level of a table onto the abdomen of an athlete lying on his back on the floor, the jumper is hurled upward by the powerful contraction of the abdominal muscles of the athlete. This is nothing but a very strong abdominal muscle reflex elicited by a stimulus applied to the abdominal muscles in their course between the costal margin and the pelvis. The best methods by which the abdominal muscle reflex may be elicited are (1) by tapping a ruler placed on the abdomen, anterolaterally at the level of the umbilicus or anteriorly with downward pressure, and (2) by tapping the finger placed above the symphysis ossium pubis.

The abdominal muscle reflex is a normal reflex, but it may often be very weak and sometimes hardly discernible, or, to all appearances, absent. Therefore, no particular significance should be attached to the apparent absence of the abdominal muscle reflex unless the abdominal skin reflex is absent too. In such a case, i. e., when both the abdominal skin reflex and the abdominal muscle reflex are absent, a lesion involving the corresponding dorsal segments or their outgoing or incoming pathways might be assumed. Greater significance must be attributed to exaggeration of the abdominal muscle reflex in the absence of the abdominal skin reflex, for exaggeration of this reflex on both sides would indicate a bilateral pyramidal lesion above the sixth dorsal segment. This exaggeration is sometimes distinct, even spectacular; the ruler or hand placed on the abdominal wall actually "jumps" upward on the slightest percussion. This exaggeration of the abdominal muscle reflex indicates a pyramidal lesion much earlier, better and more surely than does the loss of the abdominal skin reflex. I have seen patients with definite bilateral lesions of the pyramidal tract in whom the abdominal muscle reflex was distinctly exaggerated and yet the abdominal skin reflex was still present. It is noteworthy that after acute supranuclear lesions of the abdominal muscles both the abdominal skin reflex and the abdominal muscle reflex are lost; but the latter recovers and becomes exaggerated, while the former remains absent.

The abdominal muscle reflex is notably exaggerated in cases of amyotrophic lateral sclerosis, spastic paralysis and dorsolateral sclerosis. Examination of the abdominal muscle reflex has proved of special value in the commonly seen cases with signs of pyramidal involvement in both legs. When in such a case the abdominal skin reflex is lost and the abdominal muscle reflex is exaggerated, it may with some certainty be assumed that a lesion exists above the sixth dorsal segment. When the reflexes both of the abdominal skin and the abdominal muscles are lost, a lesion below this segment may be assumed. From these sketchy considerations the importance of examination of both types of abdominal reflexes may be deduced. My own experience has taught me that the careful notation of both skin and muscle reflexes should be included in every routine neurologic examination.

A study of the literature reveals that under different names a number of reflex phenomena have been described which are nothing but the deep abdominal muscle reflex described here. The earliest observation of this kind was apparently that of Gerhardt,[275] in 1895, in a case of spastic paralysis of the lower extremities due to an angioma of the fifth and sixth thoracic vertebrae. He noted an involuntary tension of

274. Kroll,[194] p. 192.

275. Gerhardt, D.: Ueber das Verhalten der Reflexe bei Querdurchtrennung des Rückenmarkes, Deutsche Ztschr. f. Nervenh. **6**:127, 1895.

the muscles of the abdomen and of the legs on percussion of the abdomen, but he did not evaluate this reaction further. In 1906, Hess [276] described briefly a new reflex and called it the "diaphragm reflex": when the nipple was tapped, a contraction of the diaphragm took place and was felt by the palpating finger as a drawing in of the processus ensiformis. Astwazaturow [277] rightly assumed that this reflex was nothing more than a "tendon reflex" of the upper part of the rectus abdominis muscle—in the terminology applied here, an abdominal muscle reflex.

The older textbooks mention a "periosteal reflex of the costal margin," or a "costal arch reflex." Purves-Stewart [278] used the term "costo-abdominal reflex." Its discovery is sometimes credited to Monrad-Krohn, but it was apparently first described by von Bechterew (cited by Rabinovitch [279]). The reflex is elicited by percussion of the costal border slightly inside the mamillary line; a contraction of the abdominal muscles follows. Monrad-Krohn [280] stated that in the great majority of cases, when the abdominal skin reflex is abolished as a result of a pyramidal lesion the periosteal reflex (as he called it) is brisk, sometimes even exaggerated. This, as has just been seen, is the typical behavior of the abdominal skin and muscle reflexes in the presence of a pyramidal lesion. The abdominal muscle reflex and the periosteal reflex of the costal margin are, of course, identical phenomena. Needless to say, it is not a periosteal reflex, but a muscle stretch reflex elicited by a tap on the elastic costal margin of the thorax, with stretching of the abdominal muscles. On physiologic examination of the so-called periosteal reflex of the abdominal wall, Altenburger [281] concluded that in this reflex the same mechanism is at play as in the tendon reflexes, and that on tapping the ribs not the periosteal but muscular receptors are stimulated. Duensing [282] tried to distinguish a costal margin–deep abdominal muscle reflex and a costal margin–superficial abdominal reflex, but this confused, rather than clarified, the subject. Unfortunately, in some textbooks, that of Church and Peterson, for example,[283] this abdominal muscle reflex is confounded with the abdominal skin reflex.

In 1908 Forster [284] mentioned briefly the elicitation of the reflex of the abdominal wall by a tap on the os pubis, but he found that this reflex was not constant. In 1923 Guillain and Alajouanine [285] described a *réflexe médio-pubien*, which attracted much attention. Purves-Stewart [278] called it "pubic tendon reflex." Guillain and Alajouanine stated that it was a bone reflex which had never been described before. The reflex consists of contraction of the abdominal muscles and of the adductors of the legs on tapping of the symphysis ossium pubis with a reflex hammer. This is, of course, not a bone reflex, but a muscle stretch, or deep muscle, reflex involving simultaneously two groups of muscles: the abdominal muscles and the adductor muscles of the thigh. The stimulus applied brings about a concussion of the symphysis ossium pubis, where both muscles have their attachment. This reflex is simply one more of the numerous instances in which more than one reflex may be elicited on one tap of a bone. What Guillain and Alajouanine called the "upper response" is simply the abdominal muscle reflex elicited by stretching of the abdominal muscles at their point of insertion, just as the old "periosteal reflex of the costal margin" is elicited from the point of origin of these muscles at the costal margin. Monrad-Krohn [280] assumed that the reflex of the costal margin and the Guillain-Alajouanine reflex are *probablement de même ordre*. The two reflexes are, in fact, one and the same, elicited only from different points of the same muscles.

Since both the abdominal muscles and the adductor muscles of the thigh are attached to the ramus superior ossium pubis, any concussion of this bone produces a stretching of the muscles inserted on each side. The so-called dissociation of the Guillain and Alajouanine reflex means simply that either the abdominal reflexes are absent and the adductor reflexes present or vice versa. This fact could be stated plainly without resorting to new names for reflexes and their dissociation.

276. Hess, O.: Ueber einen neuen Reflex, Neurol. Centralbl. **10**:485, 1906.

277. Astwazaturow: Eine biologische Deutung des Verhaltens der Reflexe bei Pyramidenbahnerkankung, Ztschr. f. d. ges. Neurol. u. Psychiat. **100**:70, 1925.

278. Purves-Stewart,[108] p. 536.

279. Rabinovitch, J. S.: Sur les réflexes abdominaux percutoires, Rev. neurol. **63**:684, 1935.

280. Monrad-Krohn, G. H.: Les réflexes abdominaux dans la sclérose en plaques, Rev. neurol. **1**:707, 1924; Reflexes of Different Order Elicitable from the Abdominal Region, Arch. Neurol. & Psychiat. **13**:750 (June) 1925.

281. Altenburger, H.: Elektrodiagnostik, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1937, vol. 3, p. 916.

282. Duensing, F.: Zur normalen und pathologischen Physiologie der Bauchdeckenreflexe, Ztschr. f. d. ges. Neurol. u. Psychiat. **168**:171, 1940.

283. Church, A., and Peterson, F.: Nervous and Mental Diseases, ed. 8, Philadelphia, W. B. Saunders Company, 1914, p. 35.

284. Forster, E.: Ueber die Bauchdecken- und Adduktorenreflexe, (*a*) Folia neuro-biol. **1**:756, 1908; (*b*) Neurol. Centralbl. **27**:231, 1908.

285. Guillain, G., and Alajouanine, T.: De réflexe médio-pubien, Compt. rend. Soc. de biol. **89**:874, 1923.

With extensive bilateral lesions of the pyramidal tract, such as those in amyotrophic lateral sclerosis, tapping of the abdominal muscles for elicitation of the abdominal muscle reflex may produce a triple response: a contraction of the abdominal muscles, a contraction of the adductor muscles and a contraction of the pectoral muscles with adduction of the arms. This triple response is easily understood. When the abdominal muscles are tapped and they contract, the points of insertion of both ends of the muscles, the thorax and the pelvis, are jarred. This jarring means a concussion and stretching of the muscles inserted there—the adductor and the pectoralis muscles—and produces the corresponding reflexes. I wonder why this triple reflex, which might well be called a pectoroabdominoadductor reflex, has not yet been described (or has it?), with all its modifications and dissociations! Just as there is no reason to attach new names to old reflexes simply because a different point of the muscle is tapped, so there is no reason for the creation of new reflexes simply on the basis that more than one reflex may be elicited by one tap on a bone or a muscle. Should this continue to be done, the number of "new" reflexes would increase ad infinitum.

Trömner,[286] perhaps more than any one else, has contributed to the classification of the reflexes and to a clear distinction between abdominal muscle and abdominal skin reflexes, but his work, written in brief, apodictic style, remained almost completely unnoticed. Trömner, by the way, was not correct in stating that the abdominal muscle reflexes were unknown until he introduced the method of their elicitation by cross percussion of the muscles. The reflexes mentioned in the foregoing discussion are all abdominal muscle reflexes. In 1926 Galant[287] described a spinoiliac-abdominal reflex and gave it the name "Galant reflex" (!). To elicit this reflex, the spina iliaca anterior superior is tapped with a percussion hammer. The reflexive movement consists of unilateral or bilateral contraction of the muscles of the abdominal wall. Galant observed it in 77.5 per cent of normal persons and concluded that it was a periosteal, and not a tendon, reflex. He noted the antagonism between the superficial abdominal reflex and the reflex he described, but could not explain it. It escaped him completely that his reflex was simply a deep abdominal muscle reflex, which in his case was elicited from the spina iliaca anterior superior, where the abdominal muscles are inserted. It is, of course, possible to elicit the stretch reflex of these muscles from any other point of their origin or insertion.

Trioumphoff[288] in 1927, at the suggestion of Astwazaturow, studied what he called the "tendon reflex of the abdomen." He obtained this reflex by tapping the region of the tuberculum pubis or of Poupart's ligament and found it as constant as the abdominal skin reflex. He noted that the reflex was absent with lesions of the spinal cord, from the sixth to the twelfth dorsal segment, and was present in cases of multiple sclerosis, when the abdominal skin reflex might be absent. In cases of advanced multiple sclerosis this reflex disappears.

Olivecrona[278] observed that the abdominal skin reflex and the abdominal muscle reflex behaved differently in cases of peritonitis—the former was lost and the latter remained normal. Byrnes,[289] in discussing the abdominal skin reflex, presented a figure that illustrates well some, but not all, of the points from which the abdominal muscle reflex can be elicited by percussion. Aronovitch[69] found that in apes the abdominal reflex, as described by Guillain and Alajouanine and by Trioumphoff, is absent but that the periosteal reflex of costal origin is present.

The phenomenon described, superficially, by Dosužkov and Bodlák,[290] in 1929, as the abdominal tendon reflex, and by Rabinovitch,[279] in 1935, as the percussion abdominal reflex, is certainly the same as the abdominal muscle reflex discussed here. Rabinovitch observed that this reflex was constant in normal adults. It was absent more or less constantly in cases of diseases of the abdominal viscera accompanied by abdominal distention or *defense musculaire* and on the site of the palsy in cases of hemiplegia. He noted further that this reflex was absent in cases of spastic paraplegia, syringomyelia and multiple sclerosis; he assumed that there is a parallelism between the skin and the percussion abdominal reflex and that for the presence of these reflexes normal function of the pyramidal tracts is necessary. My experience does not substantiate this view. A much more detailed study must be made, however, before the role

286. Trömner, E.: Zur Physiologie und Pathologie der Muskel- und Hautreflexe, Deutsche Ztschr. f. Nervenh. **77**:189, 1923; footnote 113.

287. Galant, J. S.: Die Neurologie der Bauchwand (nach Söderbergh) und der spino-iliacale Bauchreflex (Galant), Deutsche Ztschr. f. Nervenh. **90**:262, 1926.

288. Trioumphoff: Les réflexes tendineux de l'abdomen, Rev. neurol. **1**:307, 1927.

289. Byrnes, C. M.: The Abdominal Reflexes, South. M. J. **22**:260, 1929.

290. Dosužkov, T., and Bodlák, F.: The Abdominal Tendon Reflex (R.A.T.) and Its Behavior in Pyramidal Lesions with Flexion Contracture, J. Nerv. & Ment. Dis. **70**:23, 1929.

of the abdominal muscle reflex in clinical symptomatology is completely clarified. There is no uniform behavior for this reflex in such diseases as spastic paralysis, syringomyelia and multiple sclerosis. Everything depends on the localization of the morbid process. But there is hardly any doubt that the dissociation of the abdominal skin reflex and the abdominal muscle reflex—the abdominal skin reflex being absent and the abdominal muscle reflex exaggerated—is a dependable sign of a pyramidal lesion above the sixth dorsal segment.

In his article, Rabinovitch mentioned briefly the genitoabdominal reflex of Raïmiste, in which a contraction of the muscles of the abdominal wall can be obtained on percussion of the symphysis pubis. Such a reflex certainly belongs to the category under discussion. It was impossible to obtain the original article of Raïmiste, which apparently was published in Russian.

The reflex described by Weingrow,[291] in 1936, as the "external oblique abdominal tendon reflex," elicited by tapping the anterior and outer parts of the lower thoracic wall, and consisting of an outward movement of the abdominal muscles, is nothing else than the abdominal muscle reflex and is, of course, not a "tendon" reflex. It is not simply "related," as Weingrow assumed, to the periosteal reflex of the costal margin but is identical with it.

In his classic monograph on the symptomatology of diseases of the spinal cord, Foerster [292] on several occasions mentioned briefly the different behavior of the two kinds of abdominal reflexes, which he called the skin and the deep, or periosteal, reflex. Sometimes he called the latter the "osseous abdominal reflex." Nippert,[293] Foerster's pupil, differentiated an abdominal skin reflex and an abdominal periosteal reflex. He further distinguished an upper abdominal periosteal reflex, elicited by tapping the periosteum of the seventh and eighth ribs, and a lower abdominal periosteal reflex, elicited by tapping the spina iliaca anterior superior. He stated that the abdominal periosteal reflex is usually more constant than the abdominal skin reflex; after operation on the cortex the former returns to normal earlier than the latter. Kastein [294] mentioned the dissociation of the abdominal skin reflex and the abdominal muscle reflex in cases of lesions of the pyramidal tract.

Nielsen,[267] in 1940, commented on an interesting observation. He repeatedly found that the abdominal muscle reflex was increased (when the superficial abdominal reflex was absent) by inserting a finger in the umbilicus, stretching laterally, upward or downward against the muscle and then tapping the finger with a percussion hammer. This is a new, ingenious and practical way of eliciting the abdominal muscle reflex and proves, again, the multiplicity of methods by which this reflex may be elicited. In 1942 Benedek and von Angyal [295] noted in cases of multiple sclerosis a contraction of the pyramidalis muscle on percussion of the symphysis ossium pubis and called it the pyramidalis reflex. They concluded that the appearance of this reflex was a sign of a lesion in the pyramidal tract. One can only agree with Reese,[296] who stated that this reflex falls into the group of the abdominal muscle stretch reflexes under discussion. Its discovery shows, again, that the methods of elicitation of the abdominal muscle reflexes are far from being exhausted.

Despite these numerous, though scattered, observations, it is amazing to note how little is known in general about the abdominal muscle reflex, its differentiation from the abdominal skin reflex and its diagnostic importance. In his monumental work on neurology,[139] Wilson, with his awe-inspiring command of old and new neurologic literature, nowhere mentioned the abdominal muscle reflex; he referred only to the "abdominals," by which he meant the superficial reflex. Neither is the abdominal muscle reflex mentioned in many a leading textbook of neurology, old or new.

Thus, it has become almost customary to understand that the term "abdominal reflex" indicates the abdominal skin reflex only. The thesis of Frei,[297] devoted to the abdominal reflexes, begins with the sentence: "The reflexes of the abdominal wall are skin reflexes."

What is known thus far about the abdominal muscle reflex warrants the assumption that this reflex deserves its place in every textbook beside the abdominal skin reflex. Examination for the former belongs, too, in every routine neurologic examination—all the more since, in my experience, exaggeration of the abdominal

291. Weingrow, S. M.: The External Oblique Abdominal Tendon Reflex, M. Rec. **144**:317, 1936.

292. Foerster,[65] vol. 5.

293. Nippert, E.: Ueber das Verhalten der Bauchreflexe, des Hautbauchreflexes, und des periostalen, bei einzelnen Nervenkrankheiten, Dissert., Breslau, 1927.

294. Kastein, G. W.: Die Hautreflexe der Bauchwandmuskulatur, Nervenarzt **10**:85, 1937.

295. Benedek, L., and von Angyal, L.: Ueber den Musculus pyramidalis-Reflex, Monatschr. f. Psychiat. u. Neurol. **105**:109, 1942.

296. Reese, H. H.; Lewis, N. D. C., and Sevringhaus, E. L.: 1942 Year Book Neurology, Psychiatry and Endocrinology, Chicago, The Year Book Publishers, Inc., 1943, p. 184.

297. Frei, J.: Ueber die physiologische und pathologische Bedeutung des Abdominalreflexes, Dissert. [Basel], Sursee, J. Küngs Erben, 1929.

muscle reflex constitutes a fine and reliable diagnostic sign of supranuclear involvement of the abdominal muscles. But one should abandon such misnomers as periosteal, tendon, percussion and abdominal reflex; one should abandon, also, the practice of naming this reflex according to the points from which stretching of the muscle may be effected and thus confine oneself to a single name—abdominal muscle reflex. Physiologicaly, all forms of this reflex become apparent through the same mechanical excitation of the muscular tissue, of the intramuscular nerve endings, which is produced by concussion and stretching of the muscles.

QUADRICEPS REFLEX

The term "quadriceps reflex," which was used by Strümpell [171] as far back as 1879, conveys more than the designation "knee phenomenon" (Westphal), "patellar tendon reflex" (Erb), "knee jerk" (Gowers) or "knee kick." The term "knee jerk," used to designate an extensor movement of the leg, can practically be applied not only to this response but, for instance, to a reflexive bending of the knee. How confusing the term "knee jerk" may be is shown in a paper by Hinsdale and Taylor.[298] By this term the authors actually designated not the extension of the knee but the jerk of the knee in a medial direction; the phenomenon is really a cross adductor reflex. The quadriceps reflex may be elicited in different ways: First, the examiner may tap the patellar tendon, a technic which brought to this reflex the time-honored name "patellar tendon reflex," and, second, he may place his finger in a crooked position above the upper edge of the patella, press the patella downward, thus stretching the quadriceps muscle, and tap the finger (Gowers [14]). The reflex may be elicited, further, by tapping the point of origin of the rectus femoris muscle on the spina iliaca anterior inferior and finally, by broad percussion of the muscle itself. It may be mentioned that in the presence of reflexive hyperirritability, this reflex may be obtained by tapping any point of the tibia, the heel and even the posterior surface of the lower portion of the leg, but these methods are of little practical importance. It is understandable, said Tschlenow,[299] that if the hyperreflexia is outspoken, the quadriceps reflex can be elicited on percussion of any point of the lower extremity. But it is always a stretching of the muscle which is the *conditio sine qua non* for the appearance of the reflex; any tapping of the lower part of the leg, such as the tibia, is without effect if, for instance, the leg is hyperextended. Needless to say, the possibility of elicitation of the quadriceps reflex from distant points, such as the sole of the foot (Cohn,[300] Loewy [301]), designates nothing more than exaggeration of this reflex, but by no means does it in itself indicate a pyramidal lesion (Minor, cited by Tschlenow [299]). Senise [302] tried to distinguish three reflexes elicited by tapping the patella: a central, an internal marginal and an external marginal reflex. These reflexes correspond to the different portions of the quadriceps muscle which are innervated by different lumbar roots. It remains to be seen whether this subtle differentiation is of any value. Elsberg [303] spoke of the "suprapatellar, patellar and achilles reflexes." Wechsler [304] discussed, first, "the patellar or knee jerk" and, later, in a separate paragraph, the "suprapatellar reflex." He added that the reflex arc is the same as that for the patellar reflex. No wonder, since it is the same reflex! This distinction between the suprapatellar and the patellar reflex on the basis of the mode of elicitation, that is, whether the patella is moved from below or from above, has no right to exist.

In cases of pyramidal lesion, tapping of the patellar tendon may evoke not only contraction of the quadriceps muscle but, simultaneously, contraction of the plantar flexors of the foot and of the toes, even sometimes of the extensors of the hip. Thus, a mass movement, a coordinated stretch reflex, may result. This is not a deep muscle reflex but a reflex of spinal automatism. The rarely seen contralateral quadriceps reflex (Sternberg [8] and Léri [305]) is apparently only a part of such a coordinated mass movement of spinal automatism, a part of a crossed extensor reflex.

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298. Hinsdale, G., and Taylor, J. M.: Crossed Knee-Jerk, Tr. Am. Neurol. A. **20**:135, 1894.

299. Tschlenow, L.: Ueber die Korrelation der tiefen Reflexe an den unteren Extremitäten, Deutsche Ztschr. f. Nervenh. **88**:56, 1926.

300. Cohn, T.: Reflektorische Kniestreckung bei Beklopfen der Fussohle, Neurol. Centralbl. **30**:1107, 1911.

301. Loewy, E.: Die plantare Reflexzone für den M. quadriceps, Neurol. Centralbl. **32**:90, 1913.

302. Senise, T.: Essenza e valore del riflesso mesorotuleo, Cervello **13**:139, 1934; Il riflesso mesorotuleo, Riv. di pat. nerv. **43**:89, 1934.

303. Elsberg, C. A.: Surgical Diseases of the Spinal Cord, Membranes, and Nerve Roots, New York, Paul B. Hoeber, Inc., 1941, p. 383.

304. Wechsler, I. S.: A Textbook of Clinical Neurology, ed. 5, Philadelphia, W. B. Saunders Company, 1943, p. 18.

305. Léri, A., in Gilbert, A. N., and Carnot, P.: Nouveau traité de médecine et de thérapeutique, Paris, J.-B. Baillière et fils, 1926, vol. 31, p. 345.

STUDIES IN REFLEXES

HISTORY, PHYSIOLOGY, SYNTHESIS AND NOMENCLATURE: STUDY III

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ADDUCTOR REFLEX OF THE THIGH

This is a reflex of the adductor muscles of the leg. It is a sensitive and important reflex but has been somewhat neglected in favor of its great neighbor, the quadriceps reflex. The adductor muscles extend from the pelvis to the leg, and anything that brings about a concussion of either of these bony structures, so that the distance between the points of origin and the points of insertion of the abductors is increased, produces the adductor reflex.

To elicit the homolateral adductor reflex, it is best for the examiner to place his finger on the medial condyle of the femur, the leg being slightly abducted, and tap the finger, thus causing a further abduction of the leg. Any point along the inner side of the leg, including the region of the malleolus internus, or even the achilles tendon, may be tapped. The more exaggerated the reflex, the greater the distance from which it may be elicited (Keller [306]). The homolateral adductor reflex may also be elicited by a blow on the sole, or, better, on the heel, while the patient is in a prone position, or by a tap on the patellar tendon, with the patient in a sitting position, as for elicitation of the quadriceps reflex. With the last two methods, the blow on the heel or the patellar tendon may at the same time cause slight abduction of the leg and thus elict the abductor reflex directly. However, with a blow on the heel while the leg is extended or with a blow on the patella while the knee is flexed, concussion can be transmitted directly to the pelvis through the leg or the thigh respectively. The concussion of the pelvis thus produced jars the adductor muscles and elicits the adductor reflex. Even a slight concussion suffices, since the threshold for this reflex is low. Such a concussion of the pelvis can of course be effected from many other points. Tanfani,[307] in 1926, reported on the elicitation of the adductor reflex by a tap on the trochanter major. This was known long before Tanfani and was mentioned by Bickel [308] in 1912. In 1942 Grigorescu [308a] rediscovered this method of elicitation of the adductor reflex, concluded that it was a bone-tendon reflex and called it the trochanter reflex. Whether the concussion of the pelvis itself or the indirect stretching of the adductor muscles is responsible for the appearance of the reflex is a moot question.

The adductor reflex may be elicited bilaterally by tapping the symphysis pubis or the ramus superior ossis pubis (Forster [284a]) as for elicitation of the mediopubic reflex of Guillain and Alajouanine [285] or of the pubic reflex of Golomba.[309] The so-called lower response of the reflex of Guillain and Alajouanine is nothing but the adductor reflex. The adductor reflex when exaggerated can be elicited bilaterally on tapping the abdominal wall, as for elicitation of the abdominal muscle reflex. By this method, the pubic arch of the pelvis, on which the adductor muscles originate, is jarred indirectly. In lean persons the tilting effect of percussion of the abdominal muscles on the pelvis can be seen distinctly. In a case of unilateral lesion of the pyramidal tract these methods of elicitation of the adductor reflex may be used for comparison of its strength on the two sides. If the pyramidal lesion is unilateral, the adductor reflex on the affected side is stronger, or the reflex appears on mild stimulation only on this side. This difference may constitute one of the finest signs of a pyramidal lesion.

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Dedicated, in deep gratitude, to Dr. Langley Porter, dean of the University of California Medical School, 1927 to 1941.

This article is the last of a series the first and second of which have been published in the ARCHIVES (**51**:113 [Feb]; this issue, p. 341).

306. Keller, K.: Die Bedeutung des Adduktorenreflexes, Deutsche Ztschr. f. Nervenh. **37**:49, 1909.

307. Tanfani, G.: Sull'allargamento al trocantere dell' area focale reflessogena degli adduttori, Gior. di psichiat. clin. e tecn. manic. **54**:51, 1926.

308. Bickel,[70] p. 206.

308a. Grigorescu, D.: The Trochanter Reflex, Spitalul **62**:98, 1942; abstracted, Zentralbl. f. d. ges. Neurol. u. Psychiat. **103**:387, 1943.

309. Golomba, M.: Pubic Reflex, Vrach. delo **12**: 1569, 1929.

The concussion of the pelvis for elicitation of the adductor reflex may be achieved not only through the leg but through the vertebral column. Thus, a bilateral adductor reflex may be elicited on percussion of the spine.

In 1929, in a preliminary note, which was not followed by any further work, McCarthy [310] described a "spino-adductor reflex." It consisted of a sharp contraction of the adductors of both thighs on percussion of the spine. This phenomenon appeared in normal persons while sitting, but not in the upright position, on percussion of the lumbar, dorsal or cervical portion of the spine. The more exaggerated the reflex, the higher the point on the spine from which it could be elicited, even as high as the sixth cervical segment. The reflex was observed to be exaggerated in cases of pyramidal lesion and to be absent in cases of tabes. Percussion over the sacrum, the crest of the ilium and the anterior superior spine gave negative results, according to McCarthy. This is not in accordance with the observations of other investigators. Before McCarthy's report, Tschlenow,[311] in 1925, described exactly the same reflex as the "dorsal reflex of the adductors." He could elicit this reflex in cases of general reflexive hyperirritability on tapping the spinal column in the middle or paravertebrally. Kroll [312] found this reflex a valuable sign and spoke of the "Tschlenow reflex." Valobra and Bertolotti,[189] in 1905, noted that the adductors may occasionally contract when the spinal column is tapped in the lumbar or the sacral region. Von Bechterew [313] reported briefly in 1902 that he sometimes observed in sitting patients a contraction of the adductors of the thigh on tapping the lumbar vertebrae. McCarthy [310] did not cite from the literature, but in an article written twenty-five years before, he [314] mentioned the observation of von Bechterew which in every way represented his "spino-adductor reflex." Strümpell,[171] in 1879, noted a reflexive contraction of the adductors of the legs when the spinal processes of the lumbar vertebrae were tapped. Before Strümpell, Erb [315] observed, in 1875, an "outspoken reflexive contraction in the adductors on stronger tapping of the lumbar region on each side of the spinal column." There is a complete analogy to this "spino-adductor reflex" in the bilateral contraction of the shoulder muscles and the trapezius, deltoid and triceps muscles on tapping of the cervical or the thoracic portion of the spine (Sternberg [8]).

From all the points, even the distant ones, from which a homolateral adductor reflex can be elicited, such as the internal condyle, the internal malleolus, the achilles tendon and the spine and crest of the ilium, the contralateral reflex can also be elicited. It is remarkable that this contralateral reflex is often more pronounced than the homolateral one, especially when the sole is tapped. Owing to the elasticity of the pelvis, every direct or indirect concussion of this structure transmits the wave of concussion to the opposite side as a counterblow. Balduzzi,[316] in 1926, described a "new contralateral reflex of the adductor muscles." It consisted of adduction and inward rotation of the contralateral leg when the sole was tapped. Occasionally the homolateral adductors contracted reflexively, too, but this contraction was always weaker than that of the contralateral muscles. This reflex could be seen in normal persons, but only when the reflexes were lively. It was constant in cases of pyramidal lesions. This reflex is not new. Valobra and Bertolotti,[189] in 1905, reported that in 40 per cent of normal persons one can evoke a reflexive contraction of the adductors of the contralateral leg on tapping the heel.

Rasdolsky [317] described in 1923 a "crossed spinoadductor reflex," which he observed in 98 per cent of normal persons. The reflex consisted of contraction of the "crossed" (contralateral) adductor muscles when the anterior superior spine of the ilium was tapped. The homolateral adductors contracted only in 20 to 25 per cent, and less strongly. Brockaert,[318] in 1929, described the same reflex as the "crossed reflex of the pelvis"; to his knowledge, this reflex had never been described before. Myerson [169] mentioned in 1912 that a contralateral adductor response from a tap on the anterior superior spine was pointed out to him by Graves. Bickel,[308] in 1912, mentioned elicitation of an adductor reflex on tapping of the crest or the anterior superior

310. McCarthy, D. J.: The Spino-Adductor Reflex, Arch. Neurol. & Psychiat. **21**:1318 (June) 1929.

311. Tschlenow, L.: On the Dorsal Reflex of the Adductors, Russk. klin. **4**:391, 1925.

312. Kroll,[194] p. 181.

313. von Bechterew, W.: Ueber den Lumbofemoralreflex, Neurol. Centralbl. **21**:836, 1902.

314. McCarthy, D. J.: Ein neues spino-musculäres Phänomen bei normalen Personen, Neurol. Centralbl. **23**:16, 1904.

315. Erb, W.: Ueber Sehnenreflexe, Arch. f. Psychiat. **5**:792, 1875.

316. Balduzzi, O.: Un nouveau réflexe contralateral des muscles adducteurs, Rev. neurol. **2**:243, 1926.

317. Rasdolsky, I.: Der gekreuzte spino-adductorische Reflex, Arch. f. d. ges. Physiol. **202**:658, 1924.

318. Brockaert, A.: Le réflexe croisé du bassin, Presse méd. **37**:1440, 1929.

spine of the ilium. Bing [319] and Kroll,[320] in their textbooks, distinguished an adductor reflex and a spinoadductor reflex. Kroll gave the localization of the adductor reflex as the second and third lumbar segments, and Bing, as the second and fourth lumbar segments, and that of the spinoadductor reflex as the twelfth dorsal and the first lumbar segment. This statement is incorrect since the two reflexes are one and the same. The physiology of elicitation of a crossed spinoadductor reflex when the spina iliaca anterior (or posterior) superior is tapped is easily understood. The tap rotates the pelvis, or the concussion is transmitted to the opposite side in contrecoup manner. In any case, the concussion of the bone with stretching of the adductors elicits the reflex.

In the neurologic literature, such a crossed adductor reflex elicited from the patella while one taps its tendon for the patellar reflex plays a conspicuous role. The discovery of this crossed adductor reflex is usually attributed to Marie [321] and carries his name. It seems so important that the French textbooks even note the date of its discovery, April 13, 1894. Hinsdale and Taylor [298] described this reflex independently in the same year. Crocq [322] referred to Thul, who first described, in 1888, elicitation of the contralateral adductor reflex on tapping of the patella for the patellar reflex. Strümpell,[171] in 1879, mentioned the contraction of the contralateral adductors on tapping of the patellar tendon, as did Berger, [227] before him. It has been repeatedly claimed by leading neurologists that such a crossed adductor reflex constitutes a sign of pyramidal lesion (Lewandowsky,[323] Myerson,[324] Sahli [325] and Rittgerodt [326]). Lewandowsky stated that this reflex was one of the most valuable pyramidal signs; others compared it to the Babinski and Oppenheim signs. My own experience has taught me that the crossed adductor reflex is an entirely normal phenomenon. It can be elicited from many points of the contralateral leg, and often much more easily than the homolateral adductor reflex. It is, of course, exaggerated on the side of a pyramidal lesion, as are all other reflexes, but it must be stressed that the crossed adductor reflex appears in normal persons and in itself does not constitute a sign of pyramidal involvement. Reference may be made to the same or to similar views expressed by Hinsdale and Taylor,[298] Ganault,[327] Graeffner,[328] Marie,[321] Cacciapuotti,[329] Kastein,[226] Stern,[203] Wegener,[330] Balduzzi [316] and Foix and Chavany.[331]

Authors who see in the adductor reflex a pathologic sign usually explain its appearance through transmission of applied stimuli via the spinal cord. Valobra and Bertolotti,[189] for instance, stated that the vibration of the bony structure of the lower extremities causes an irritation of the posterior roots of the spinal cord, where the reflex originates. They were unable to explain why this irritation of the posterior roots preferably manifests itself in the movement of the adductors. There has been considerable controversy as to whether the contralateral reflexes are transmitted peripherally or centrally. A contralateral adductor reflex may, it is true, be a manifestation, a part, of a crossed extensor reflex, and may thus belong to the so-called reflexes of spinal automatism. But it is seldom of this nature. In the majority of cases, especially those of normal persons, general considerations and clinical observations speak in favor of a purely peripheral, mechanical transmission. Through concussion of the bone, a simple muscle stretch reflex is evoked on the opposite side.

A deep muscle reflex is a completely isolated entity. A stimulus evoking such a reflex does not invade other fields. The reflex is, so to speak, a private affair of the corresponding muscle. To assume that on tapping the patella, one evokes a contralateral adductor reflex by radiation of the stimulus in the spinal cord would be assuming something that has no analogy in any other muscle. In the upper extremity, for instance, all the reflexes which may unavoidably be evoked simultaneously can easily be explained on a peripheral, mechanical basis. When, on elicitation of a deep reflex of a single muscle, other muscles than those that might be

319. Bing, R.: Compendium of Regional Diagnosis, St. Louis, C. V. Mosby Company, 1940, p. 87.

320. Kroll,[194] p. 180.

321. Marie, P.: Sur la contraction réflexe des adducteurs de la cuisse, Bull. et mém. Soc. méd. d. hôp. de Paris **11**:246, 1894; Rev. neurol. **2**:399, 1894.

322. Crocq, J.: Physiologie et pathologie du tonus musculaire, J. de neurol. **6**:424, 1901.

323. Lewandowsky,[80] p. 598.

324. Myerson, G.: The Value and Meaning of the Adductor Responses of the Leg, J. Nerv. & Ment. Dis. **43**:121, 1916.

325. Sahli,[135] p. 223.

326. Rittgerodt, W.: Ueber die gekreuzten Adductorenreflexe, Dissert., Cologne, 1936.

327. Ganault, E.: Contribution a l'étude de quelques réflexes dans l'hémiplégie d'origine organique, Thesis, Paris, no. 542, 1898.

328. Graeffner: Einige Studien über Reflexe, München. med. Wchnschr. **1**:489, 1908.

329. Cacciapuoti, G.: La reazione controlaterale alla percussione rotulea o mesorotulea, Cervello **13**:248, 1934.

330. Wegener, H.: Der gekreutze Adduktorenreflex, Monatschr. f. Psychiat. u. Neurol. **66**:342, 1927.

331. Foix and Chavany,[188] p. 572.

stretched indirectly by the concussion respond simultaneously, it must be assumed, first, that not the deep muscle reflexes, but associated movements, manifestations of spinal automatism, are brought into play. Rasdolsky,[317] pleading the spinal origin of his crossed spinoadductor reflex through radiation of the stimulus in the spinal cord, offered what he thought convincing proof. A man had a lesion of the cervical part of the spinal cord with spastic paralysis of the extremities, especially of the upper part of the right arm. In this case, tapping of the patellar ligament and of the superior iliac spine on the left side evoked not only different reflexes in the lower extremity, but pronounced contraction of the muscles of the upper extremity on the right side. This, of course, was not a deep muscle reflex but a clear-cut associated movement due to spinal automatism. Many sensory stimuli other than tapping of the patella and the iliac spine, and many other manipulations could have produced this associated movement of the spastic upper extremity.

The peripheral, mechanical origin of the contralateral adductor reflex is plainly evident on careful clinical observation. When a patient is in a sitting position, a strong blow on the patella may act on the pelvis in different ways: 1. The blow may, through the femur, be transmitted directly to the pelvis, longitudinally. 2. The blow, in mild degree, may abduct the thigh directly and thus jar the pelvis through the homolateral adductors. 3. The homolateral patellar and adductor reflexes, which may be simultaneously elicited, may jar the pelvis through contraction of the corresponding muscles, which originate on the pelvis. In lean persons this jarring of the pelvis can be seen easily on examination for the quadriceps reflex, especially when the patella is pushed down and tapped on its upper border. From his experience with experimental animals, Sherrington,[332] stated:

> It is remarkable how distinctly even a slight tap upon the end of femur is felt by holding the lower end of the other femur, particularly in some positions of the thigh, especially, I think, when the thighs are somewhat abducted.

That the elastic structure of the pelvis transmits a greater wave of concussion to the opposite side is easily understood. This condition also obtains in the chest. In the "contralateral periosteal reflexes of the arm," described by Myerson[173] in 1914, tapping of the middle of the clavicle on one side produces a contraction of the biceps and pectoralis major muscles on the other side. Strümpell[171] and Westphal[333] had reported the same phenomenon previously. The following simple demonstration will prove the transmission of the stimulus via the pelvis. The crossed adductor reflex appears in a patient in a sitting position when the patella is tapped, as for elicitation of the quadriceps reflex; the blow is directed toward the pelvis and transmitted through the femur. If the same patient is prone when the patella is tapped, i.e., if the blow is directed not toward the pelvis, but downward and vertically, toward the femur, the crossed adductor reflex cannot be elicited with a blow of the same strength. Unfortunately, in reports on the crossed adductor reflex the position of the patient is usually not mentioned; whenever it is, the reference is to a sitting position. The same is true of the crossed adductor reflex elicited by a blow on the heel. It is possible to elicit this reflex when the patient is lying down with the leg extended. Here the blow can be transmitted directly to the pelvis. However, the crossed reflex does not appear when the leg is bent at the hip and at the knee joint. Here the blow on the heel is not directed toward the pelvis. The following case offers a significant illustration of the importance of the mechanical factors involved in elicitation of the crossed adductor reflex. In a middle-aged woman with intact nervous system, the crossed adductor reflex could be elicited when the patient was lying down, with the legs extended, by tapping the right heel only. The reason was that she had complete arthrodesis of the right knee. The blow on the right heel could more easily be transmitted to the pelvis than a blow on the left heel, since on the left side the normal knee joint acted as a cushion and absorbed part of the blow, while the arthrodesis on the right side facilitated the transmission of the blow to the pelvis. That the mechanical blow, and not the elicitation of the patellar reflex, produces the crossed adductor reflex was shown by Russell,[334] who emphasized that a similar blow, such as one on the patellar tendon but delivered on the distal end of the femur, may evoke a crossed adductor reflex. Schäffer[335] found that the latent period for the contralateral reflex was just as long as that for the homolateral reflex. This observa-

332. Denny-Brown, D.: Selected Writings of Sir Charles Sherrington, New York, Paul B. Hoeber, Inc., 1940, p. 152.

333. Westphal: Ueber einen Fall von polyneuritischer "Korsakow'scher" Psychose mit eigenthümlichem Verhalten der Sehnenreflexe, Deutsche med. Wchnschr. **28**:81, 1902.

334. Russell, J. S.: The Crossed Knee Jerk, Am. J. M. Sc. **111**:306, 1896.

335. Schäffer, H.: Ueber Sehnenreflexe und die Methodik ihrer Latenzzeitbestimmung, Ztschr. f. d. ges. Neurol. u. Psychiat. **74**:605, 1922.

tion fits into the conception that the two reflexes are elicited by the same mechanism, which does not require any transmission to the spinal cord. Noteworthy in this connection are the experiments of Sherrington,[332] who found that contralateral reflexes persist "after complete longitudinal splitting of the cord from a lumbar transection above to the coccygeal nerves below." Schultze[336] observed that the contralateral adductor reflex was preserved in rabbits after the nervus cruralis was cut on both sides. Kornilow[337] noted in rabbits, also, that no muscles or nerves are necessary for transmission of the contralateral reflex. Only conduction through the bone is required. The idea of the purely mechanical origin of the contralateral reflexes, as advocated here, is very old. As far back as 1882, Westphal[338] espoused the same theory.

An interesting phenomenon, not easily understood and occasionally reported in the literature (Marie,[321] Strümpell,[339] Huismans,[340] Westphal,[338] Myerson,[341] Yawger[342] and Herschmann[343]), may be described as follows: When the patellar reflex is lost on one side, the tapping of the patellar tendon evokes an exaggerated adductor response on the same, or on the opposite side. When the achilles reflex is lost, the tapping of the achilles tendon produces the same result. For elicitation of this reflex, the same mechanical conditions as those previously discussed prevail, but it is quite permissible to assume—as I[344] did previously—that the reflexes show generally an increased irritability if their neighborhood reflex arcs are damaged. This is apparently an isolation phenomenon. Noteworthy is the observation of Westphal[338] in a case of alcoholic polyneuritis: The contralateral adductor reflex, obtained on tapping the patellar tendon, was lively as long as the patellar reflex was absent. When, on recovery, the patellar reflex returned, the crossed adductor reflex could not be elicited as constantly and as distinctly as when the patellar reflex was absent.

A rare phenomenon is the contralateral quadriceps reflex. Strümpell[171] mentioned having once observed a weak contraction of the contralateral quadriceps muscle on elicitation of the homolateral reflex from the surface of the tibia. Léri[305] compiled the observations of other investigators on the same phenomenon. It is hardly probable, though possible, that a tap on the patella could, via the pelvis, transmit the concussion to the opposite quadriceps muscle. It is most likely that this phenomenon is a crossed extensor reflex, of which extension of the knee is an important part. This is a manifestation of spinal automatism. Here, tapping of the patella acts merely as a stimulus to the skin; the reflexive contraction of the quadriceps muscle acts, too, as a stimulus for elicitation of the crossed extensor reflex.

BICEPS FEMORIS REFLEX

One can best elicit this reflex by tapping the muscle above its attachment to the capitulum fibulae. This is most easily accomplished with the patient lying on the side opposite the one tapped, with the leg bent at the hip and at the knee. When the tendon is tapped, its forward springing can be seen and the contraction of the muscle observed as well. Usually this is the only reaction to the tap. Occasionally the semitendinosus and semimembranosus muscles react, too, and slight bending of the knee may occur. Sometimes this muscular contraction may be felt on palpation better than it can be seen. The biceps femoris reflex is a normal one. It was constantly obtained in 350 persons by Loewenstein.[345] Its spinal center is the fifth lumbar and the first and second sacral segments. Under pathologic conditions the biceps femoris reflex behaves in the same manner as the quadriceps and the triceps surae reflex. But in cases of tabes it may disappear on one or both sides, even before the other deep reflexes have been lost (Guillain and Barré[346]). It may even be the only reflex lost

336. Schultze, F.: Bemerkungen zur Lehre von den Sehnenreflexen, Deutsche Ztschr. f. Nervenh. **78**:123, 1923.

337. Kornilow, A. A.: On the Contralateral (Crossed) Reflex, Zhur. nevropat. i psikhiat. **2**:1114, 1902.

338. Westphal, C.: Ueber eine Fehlerquelle bei Untersuchung des Kniephänomens und über dieses selbst, Arch. f. Psychiat. **12**:798, 1882.

339. Strümpell, A.: Zur Kenntniss der Haut- und Sehnenreflexe bei Nervenkranken, Deutsche Ztschr. f. Nervenh. **15**:254, 1899.

340. Huismans, L.: Gekreuzte Adduktorenreflexe bei Syringomyelie und Neuritis, Deutsche med. Wchnschr. **28**:882, 1902.

341. Myerson, A.: Note on Adductor Response to Homolateral and Contralateral Stimulation, Contrib. Psychopath. Hosp., Dept. Boston State Hosp., 1913, p. 85.

342. Yawger, N. S.: A Case Showing Exaggerated Knee Jerk on the Right and Absent Knee Jerk on the Left, with Each Side Giving a Homolateral and a Contralateral Abductor Response, J. Nerv. & Ment. Dis. **47**:285, 1918.

343. Herschmann, H.: Kontralateraler Adduktorenreflex, Wien. klin. Wchnschr. **46**:997, 1933.

344. Wartenberg, R., in discussion on Hoffmann, P.: Ueber die Ausbreitung von Reflexen und die Isolation in Reflexbahnen, abstracted, Klin. Wchnschr. **9**:1793, 1930.

345. Loewenstein, J.: Ueber den Sehnenreflex des Biceps femoris, Deutsche Ztschr. f. Nervenh. **89**:108, 1926.

346. Guillain, G., and Barré, J. A.: Abolition précoce du réflexe péronéofémoral chez un tabétique, Bull. et mém. Soc. méd. d. hôp. de Paris **41**:901, 1917.

permanently in this disease (Loewenstein [345]). Barré [347] found this reflex preserved in cases of truncal sciatica and lost in cases of funicular or radicular sciatica. Foerster [348] stressed the importance of this reflex for the diagnosis of circumscribed lesions of the upper lumbar portion of the cord and of the cauda equina. With mild lesions of the fifth lumbar segment, it may be absent while the triceps surae reflex is present. With lesions of the first to the fourth lumbar segments, the quadriceps reflex may be absent and the biceps femoris reflex exaggerated. When this reflex was exaggerated, it could be elicited by Foerster [348] not only from the tendon and the capitulum fibulae, but from the greater trochanter.

The biceps femoris reflex was described by Erb [315] and was mentioned by Sternberg [8] in his classic monograph. Guillain and Barré [349] described it as the posterior peroneofemoral reflex. They mentioned only the work of Dejerine and stated that otherwise the literature was mute on this subject. In this they were in error, as were the authors who attributed the first description of this reflex to Guillain and Barré.

REFLEX OF THE SEMITENDINOSUS AND SEMIMEMBRANOSUS MUSCLES

The reflex of the semitendinosus and semimembranosus muscles is of little clinical significance. As far back as 1879, Strümpell [171] noted a contraction of these muscles on tapping the achilles tendon. McCarthy [314] saw the contraction of these muscles on tapping the second and third lumbar vertebrae. This mode of elicitation is understandable, since the semitendinosus and semimembranosus muscles take their origin on the pelvic bone and are influenced by any percussion of the vertebral column or the pelvis, as are the adductor muscles. Noïca and Strominger,[350] in 1906, showed that reflexive contraction of the semitendinosus and semimembranosus muscles can be achieved by tapping the tibia, the malleolus internus or the crista iliaca. Guillain and Barré [351] rediscovered this reflex and gave it the name posterior tibiofemoral reflex. According to these authors, it is a tendon reflex because it can be elicited on tapping the tendon, but it is a periosteal reflex as well, since it can be elicited also on tapping the periosteum!

KNEE FLEXOR REFLEX

With the patient lying on one side, his knee bent at a right angle, a sudden, jerky stretching of the lower portion of the leg, produced, for instance, when the calcaneum is tapped in a posteroanterior direction, causes a reflexive contraction of the flexors of the leg. The practical significance of this reflex is not great. It may be absent in cases of early tabes. The name given by Ravà [352] to this reflex—periosteal reflex of the calcaneum—clouds the issue, since it is neither a periosteal reflex nor a reflex of the calcaneum, but is a deep muscle reflex of the flexors of the knee. Noïca and Strominger [350] noted a reflexive contraction of all the flexor muscles of the knee on tapping the crista iliaca. Dejerine [353] mentioned the occurrence of the same contraction when the tuberosity of the tibia was tapped. Other points of elicitation mentioned by Foerster [354] were the capitulum of the fibula, the epicondylus of the femur, the greater trochanter and the sole of the foot.

TRICEPS SURAE REFLEX

Custom and brevity would plead for the time-honored name achilles reflex, but physiologic correctness demands the term triceps surae reflex. Gowers [14] remarked, as long ago as 1880, that it is in the highest degree doubtful whether this reflex has anything to do with the achilles tendon. There are three main ways in which this reflex can be elicited. The best method of reenforcement is the same for all three: slight active plantar flexion of the foot. The first way in which one can elicit the triceps surae reflex is by tapping the achilles tendon directly. This can be done when the patient is lying down, kneeling or, best, sitting with the soles planted firmly. The second point from which the triceps surae reflex may be elicited is the planta pedis. Later, in discussion of the plantar muscle reflex, it will be seen that this reflex of the plantar flexors of the toes can be elicited by tapping the ball or the middle part of the foot. This technic is associated with the names of Zhukovski, Kornilow, and Guillain and Barré. Nearly all authors noted that this reflex consists not only of a flexion of the toes but of a plantar flexion of the foot. The latter movement is merely a triceps surae reflex

347. Barré, J. A.: La sciatique, Presse méd. **27**:57, 1919.

348. Foerster, O., in discussion on Lowenstein,[345] p. 112.

349. Guillain and Barré: Le réflexe péronéo-fémoral postérieur, Bull. et mém. Soc. méd. d. hôp. de Paris **41**: 287, 1917.

350. Noïca, D., and Strominger, L.: Réflexes osseux, Rev. neurol. **14**:969, 1906.

351. Guillain and Barré: Le réflexe tibio-fémoral postérieur, Bull. et mém. Soc. méd. d. hôp. de Paris **41**: 284, 1917.

352. Ravà, G.: Riflesso periosteo del calcagno, Riforma med. **37**:179, 1921.

353. Dejerine,[156] p. 949.

354. Foerster,[195] p. 894.

elicited by indirect stretching of the corresponding muscle. The stretching occurs through a tap on the sole, which causes sudden dorsiflexion of the foot; it is best effected with the knee bent. In general, one may more easily achieve a stretching of the triceps surae muscle, and thus elicit its reflex, by tapping the achilles tendon directly than by the indirect method of tapping the sole of the foot. In the latter case, the soft part of the foot, the elasticity of the bones of the foot, and the lever of the foot absorb a great deal of the strength of the blow before it reaches the length of the muscle. It is therefore understandable that a blow of certain strength can more easily elicit the reflex from the tendon than from the sole; if the triceps surae reflex is weak, it can be elicited only by tapping the achilles tendon. It follows that if there is an increase in the strength of the triceps surae reflex on one side, the reflex may quite possibly be elicited from the sole on this side only, and not from the sole on the normal side. Thus, this method may be of value in detecting slight differences between the reflexes on the two sides, provided of course that the orthopedic mechanical conditions of the foot for the transmission of the impulse are the same on the two sides. So considered, the plantar flexion of the foot elicitable on tapping the planta pedis is identical with the triceps surae reflex. It is, therefore, a normal phenomenon. Some time has been required for this to be realized.

Strümpell,[339] in 1899, and Cohn,[800] in 1911, mentioned casually the reflexive plantar flexion of the foot when the sole was tapped. In 1910, Andres,[355] discussing the Zhukovski reflex, emphasized that the component of this reflex, which consists of plantar flexion of the foot, is not a new reflex but a "modification of the achilles reflex." Here, again, the term "modification of a reflex" is used. To be exact, one should speak of a modification of the technic for elicitation of the reflex. The reflex itself undergoes no modification whatever. Vitek,[356] in 1905, described this reflex as a "new reflex of the planta pedis" and noted that it behaved like the patellar and the achilles reflex. But in his opinion, and in that of Lewandowsky,[357] it was not a masked achilles reflex. Although Vitek had noted the presence of this reflex in a large percentage of normal persons, Zhukovski [358] listed Vitek's reflex, together with the reflexes of Babinski, Oppenheim and Rossolimo, as having essential significance in the diagnosis of nervous diseases. Reimer [359] rediscovered this reflex, in 1920, as a normal "deep reflex of the sole of the foot." He found that the most sensitive point for elicitation of this reflex was the lateral edge of the foot proximal to the fifth metatarsal tuberosity. In his opinion, the receptor for this reflex was not the plantar fascia but the plantar aponeurosis. He distinguished this reflex from that of the achilles tendon and called it an aponeurotic reflex. Though the plantar aponeurosis, according to Reimer, is embryonically closely connected with the achilles tendon, it has assumed in man a separate form and is as independent an organ as the achilles tendon. Popper [360] called Reimer's reflex a sole tap reflex. Berger,[361] Strümpell,[362] Weigeldt [363] and others proved the complete identity of the reflexes of Vitek and Reimer with the achilles reflex. Shatz [364] emphasized the identity of the corresponding part of the Guillain-Barré reflex with the achilles reflex. Guber-Gritz and Istomin [365] did the same with regard to the corresponding part of the Zhukovski reflex. According to these authors, the plantar flexion of the foot elicited by tapping the planta pedis occurs less frequently after the age of 50, only in 16 per cent of cases. This is doubtless due to decrease in the elasticity of the joints of the foot with the advance of age. Historically, it is interesting that Sternberg [8] listed a reflex which could be evoked by tapping the sole of the foot and causing a contraction of the gastrocnemius muscle.

A third place from which the triceps surae reflex may be elicited is the lower part of the front of the leg. It has previously been mentioned, especially with regard to the so-called paradoxic triceps reflex, that it is natural to elicit a reflex not by tapping the muscle on the side of the joint on which it originates, but by tapping the joint on the opposite side, the blow having a depressing effect on the joint and thus indirectly stretching the muscle. This applies to the achilles reflex,

355. Andres, L.: Plantar-Toe Flexion Phenomenon (Zhukovski), Obozr. psikhiat., nevrol. **15**:401, 1910.

356. Vitek, V.: Ein neuer Reflex auf der Planta pedis, Neurol. Centralbl. **24**:402, 1905.

357. Lewandowsky,[80] p. 604.

358. Zhukovski, M. N.: Plantar-Toe Flexion Phenomenon, Obozr. psikhiat., nevrol. **15**:200, 1910.

359. Reimer, O.: Ein Tiefenreflex an der Fussohle, Med. Klin. **16**:848, 1920; **17**:563, 1921.

360. Popper, E.: Ein Tiefenreflex an der Fussohle, Monatschr. f. Psychiat. u. Neurol. **49**:45, 1921.

361. Berger, A., in discussion on Vitek,[356] p. 592.

362. Strümpell, A.: Ueber den Reimerschen Tiefenreflex an der Fussohle und über reflexogene Zonen, Med. Klin. **17**:97, 1921.

363. Weigeldt, W.: Der plantare Sehnenreflex ein modifizierter Achillessehnenreflex, Deutsche Ztschr. f. Nervenh. **71**:173, 1921.

364. Shatz, M.: Modification of the Achilles Reflex, Sovet. psikhonevrol. **9**:57, 1933.

365. Guber-Gritz, D. S., and Istomin, P. P.: Problem of the Sole-Toe Flexion Reflex: Phenomenon of Zhukowski, Vrach. delo **8**:740, 1926.

too. This reflex can be elicited not only from the dorsal aspect of the leg on the side of the achilles tendon, but from the frontal aspect, i. e., from the head of the tibia to the ankle joint. This is facilitated, or made possible, by passive dorsiflexion of the foot, which increases tension of the triceps surae muscle. The passive elongation of the muscle greatly increases its responsiveness to concussion and further stretching. A tap on the front of the tibia while the foot is kept in dorsiflexion stretches the achilles tendon and the triceps surae muscle. This is brought about by depression of the tibia and further dorsiflexion of the foot. When the upper part of the tibia is tapped, the foot being held in dorsiflexion, the triceps surae muscle may also be stretched by the depression of the knee joint provoked by this tap.

Gowers,[366] in 1880, introduced and illustrated a method of elicitation of the achilles tendon reflex by a "front tap." While the patient is in a sitting position, with the leg extended, the examiner holds his foot in dorsiflexion. The muscles of the leg are tapped, and a plantar flexion of the foot follows.

> . . . It is clearly the result of the passive tension placed upon the muscle, and it is, perhaps, a true reflex phenomenon, the afferent impulse being the tension on the muscular fibres.

Walton and Paul [367] devoted a special study to "the achilles-jerk and front-tap," being unaware that the two phenomena represent one and the same reflex of the plantar flexors of the foot. They observed that a reflex was produced by the frontal tap in about 40 per cent of normal persons and in 71 per cent of persons with functional disorders.

Different modes of elicitation of the triceps surae reflex, identical with the "front tap" method, have been described under various and fanciful names by neurologists who were apparently oblivious to Gowers' classic work. Piotrowski,[368] in 1913, described "a new antagonistic reflex," consisting of plantar flexion of the foot on percussion of the tibialis anticus muscle between the tuberosity of the tibia and the head of the fibula. For him, this reflex had the same importance as the pyramidal signs of Babinski, Rossolimo, Oppenheim, Mendel and von Bechterew. His view was confirmed by Stern [369] and by certain Polish authors—for a review of the literature see Sagin [370]—who regarded the Piotrowski reflex as pathognomonic of a pyramidal lesion and as being a more sensitive sign of pyramidal involvement than any other. The truth is that this reflex indicates merely an exaggeration of the triceps surae reflex. Its elicitation is greatly facilitated by stretching of the triceps surae muscle through passive dorsiflexion of the foot. The tap acts as a depressor of the knee joint, with resultant stretching of the triceps surae muscle; the tap stretches this muscle also by increasing the dorsiflexion of the foot, the more so since the foot is being already held in dorsiflexion. This reflex amounts practically to Gowers' "front tap" reflex, with the tap applied more proximally. It is certainly easier to effect a stretching of the triceps surae muscle and to elicit its reflex by tapping the achilles tendon directly than by the indirect method of depressing the joints. Therefore, the Piotrowski reflex can be elicited only when the achilles reflex is exaggerated, and it may be absent in normal persons. But by no means does it in itself indicate a pyramidal lesion. Siemionkin [371] obtained the Piotrowski reflex in some patients with schizophrenia and called it a sign of an extrapyramidal lesion!

In 1918, Bing [372] reported "a new sign of organic spasticity"—the paradoxic "ankle reflex." He mentioned this reflex again in each edition of his popular textbooks. The reflex consists of plantar flexion of the foot when any point of the line of the ankle joint between the crest of the malleolus externus and that of the malleolus internus is tapped. As Bing emphasized, it is of no significance whether the tendon of the tibialis anticus or the tendon of the extensor hallucis longus or the tendon of the extensor digitorum pedis communis muscle is tapped. An essential preliminary step for elicitation of this reflex is dorsiflexion of the foot. Bing asserted that this reflex is pathognomonic of spasticity due to an organic lesion. He stressed its close relation to the reflex of Piotrowski. The zones from which these two reflexes can be elicited are contiguous. Tschlenow [299] stated that the reflexogenous zones for Bing's reflex extend beyond the boundaries given by Bing. He could elicit Bing's reflex from a more proximal point of the frontal aspect of the lower part of the leg, even as high as the middle of the leg. This is the place at which Gowers applied his frontal

366. Gowers,[14] p. 24.

367. Walton, G. L., and Paul, W. E.: Contribution to the Study of the Achilles-Jerk and the Front-Tap, Tr. Am. Neurol. A. **29**:22, 1903.

368. Piotrowski, A.: Ueber einen neuen antagonistischen Reflex, Berl. klin. Wchnschr. **50**:726, 1913.

369. Stern, F.: Die klinische Verwertbarkeit des Piotrowskischen antagonistischen Reflexphänomens, Med. Klin. **12**:364, 1916.

370. Sagin, K.: Le réflexe de la malléole externe et le phénomène de Piotrowski, Rev. neurol. **1**:319, 1927.

371. Siemionkin, M.: Sur le caractère intérieur du phénomène de Piotrowski, Rev. neurol. **1**:1041, 1925.

372. Bing, R.: Ein neues Zeichen organisch bedingter Spastizität, Cor.-Bl. f. schweiz. Aerzte **48**:465, 1918.

tap. In my opinion, the reflex of Bing is neither paradoxic nor indicative of a pyramidal lesion. Elicitation of the achilles reflex by tapping the front of the leg is a completely normal procedure, as normal as elicitation of the biceps reflex by tapping the convex side of the elbow joint. Passive dorsiflexion of the foot greatly facilitates the further stretching of the triceps surae muscle produced by tapping the frontal aspect of the leg, including the frontal line of the ankle joint. It is hardly necessary, therefore, to elaborate on the simple mechanical fact that when the foot is in dorsiflexion, the triceps surae muscle being stretched, a sharp tap in the depth of the angle thus formed will contribute to further dorsiflexion of the foot and will thus stretch the triceps surae muscle and elicit its reflex. It is therefore not a pyramidal sign, though it can generally be elicited when the reflexes are lively or exaggerated.

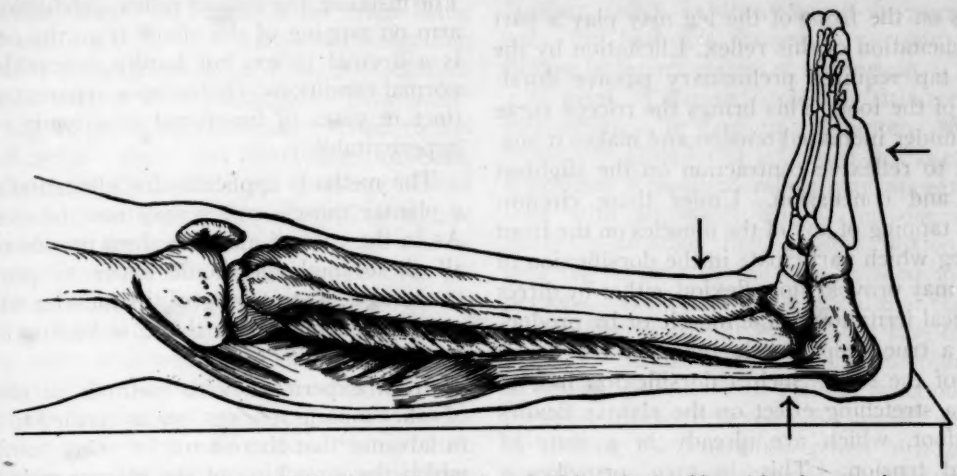

Fig. 6.—Triceps surae reflex. With the subject's foot held loosely dorsiflexed by the examiner, a tap of the reflex hammer in the direction of any of the three arrows can effect a stretching of the triceps surae muscle and thus elicit the triceps surae reflex.

In 1910, Trömner [373] mentioned briefly that one can elicit the achilles reflex from the malleolus externus. Bing,[372] in 1918, said that his reflex could even be elicited by tapping the distal parts of the malleoli. Unaware of this statement, Balduzzi,[374] in 1925, described a new reflex of the malleolus externus, which he observed in 6 per cent of normal persons. The examiner dorsiflexes the foot and taps the frontal edge of the malleolus externus. There was no doubt in Balduzzi's mind that this reflex was periosteal, since it was elicited "by mechanical irritation of the periosteum of the external malleolus." Sagin,[370] in a comparative study of the reflex of Piotrowski and that of Balduzzi, stated, rightly, that the latter is not fundamentally new, but represents only "an extension of the reflexogenous zone of the Piotrowski reflex to the malleolus externus." The same could be said of the relation of the reflexes of Balduzzi and Bing, the reflex of Balduzzi being a slight modification of the technic used by Bing. Bing [372] himself noted that his reflex could also be elicited on tapping the distal parts of the maleoli, especially in the anteroposterior direction.

Under the fanciful name "a pathologic fascia cruris reflex," Benedek [375] described a sign consisting of a plantar flexion of the foot when the foot was slightly dorsiflexed and the frontal edge of the fibula was tapped in the middle of the lower portion of the leg. He stated that this reflex should not be separated from that of Piotrowski. The mode of elicitation of the two reflexes is indeed so similar that they must be regarded as identical. The same can be said of all the reflexes under discussion that can be elicited from the frontal aspect of the leg: the "frontal tap" reflex of Gowers and the reflexes of Piotrowski, Bing, Balduzzi and Benedek. They are all essentially normal reflexes elicited in a normal way by stretching of the triceps surae muscle. They are not merely closely related phenomena; they are one and the same reflex and are distinguished only by a different point of elicitation. All the reflexes under discussion are not, as some authors, in true recognition of their nature, have claimed them to be, "modifications of the achilles reflex." The achilles reflex undergoes no modification; only the technic of its elicitation differs. These reflexes

373. Trömner, E.: Ueber einen neuen Fussreflex (Malleolarreflex), Neurol. Centralbl. **29**:528, 1910.

374. Balduzzi, O.: Der Reflex des Malleolus Externus, Monatschr. f. Psychiat. u. Neurol. **58**:31, 1925.

375. Benedek, L.: Ein pathologischer Fascia-cruris-Reflex, Deutsche Ztschr. f. Nervenh. **78**:104, 1923.

are the achilles reflex (fig. 6). The front of the leg is a large area. Therefore there are still many more points of elicitation left in this area from which "new" reflexes may be obtained.

Some authors, such as Gowers, Pietrowski and Stein,[203] who have recognized the true relation of the reflexes just discussed to the achilles reflex, have laid too much stress on the significance of tapping the tendons of the muscles, or the muscles themselves, on the front of the leg for the reflexive plantar flexion of the foot. These muscles on the front of the leg may play a part in the elicitation of this reflex. Elicitation by the frontal tap requires preliminary passive dorsiflexion of the foot. This brings the triceps surae muscle under increased tension and makes it susceptible to reflexive contraction on the slightest stretch and concussion. Under these circumstances, tapping of any of the muscles on the front of the leg which participate in the dorsiflexion of the foot may provoke dorsiflexion, either by direct mechanical irritation of the muscle or by production of a true deep muscle reflex of the dorsiflexors of the foot. Such a dorsiflexion has, of course, a stretching effect on the plantar flexors of the foot, which are already in a state of increased tension. This, in turn, provokes a reflex reaction of the plantar flexors.

Nevertheless, I wish to stress the importance of the role of the joints in the production of the "front tap" reflex. This is clearly seen in Bing's reflex, which can be elicited on tapping the malleoli, or in the reflex of Balduzzi, brought about by tapping the external malleolus. Benedek,[375] too, emphasized that his reflex could be elicited not only on percussion of the tibialis anticus muscle but on tapping of the anterior edge of the fibula. Here, the tap works not on the muscles but on the joint.

It is my experience that the triceps surae reflex can easily be elicited from the front of the leg by tapping the lower third of the tibia. It is essential to keep the leg slightly flexed at the knee while the foot is held dorsiflexed, an elastic pressure being exerted on its ball.

PLANTAR MUSCLE REFLEX

The plantar skin reflex represents the superficial, the skin-muscle reflex, of the plantar muscles in the same way as the abdominal skin reflex represents the superficial, the skin-muscle, reflex of the abdominal muscles. From a purely theoretic standpoint, it must be assumed that the plantar muscles, the flexors of the toes, should also have a muscle reflex of their own, i. e., a muscle stretch reflex, a so-called deep muscle reflex, as have all other striated muscles of the human body. It would be a striking exception to the general rule if this were not the case. It would be no exception, however, if this assumed plantar muscle reflex existed, as reflexes of some other muscles do, in latent form, becoming apparent only on application of a special technic, or only occasionally, when there is functional or organic reflexive hyperirritability. It is essential to bear in mind always that such latency of the deep muscle reflexes is common. For instance, the deltoid reflex—abduction of the arm on tapping of the elbow from the outside—is a normal reflex, but hardly noticeable under normal conditions. It becomes apparent and distinct in cases of functional or organic reflexive hyperirritability.

The methods applicable for elicitation of such a plantar muscle reflex may now be examined. As in the case of all other deep muscle reflexes, all these methods should serve to provoke a concussion, a stretching of the muscles with consequent contraction, in this case leading to bending of the toes.

From experience with methods of elicitation of other muscle reflexes, one is entitled to assume in advance that there must be many points from which the stretching of the plantar muscles and elicitation of their reflex can be accomplished.

The most natural method of elicitation of the plantar muscle reflex is, obviously, a sudden, quick tap on the plantar surface of the toes, which produces a brisk stretching of the plantar flexors. This is exactly the method for elicitation of the Rossolimo reflex, about which a vast amount of literature exists. This reflex, described by the Moscow neurologist Rossolimo[376] in 1902 and 1908, consists of sudden flexion of the toes when their plantar surfaces are lightly struck.[377] My experience is that this reflex is best elicited when, before striking the toes, the examiner brings the whole foot passively in dorsiflexion by exerting pressure on the instep, thus stretching the plantar muscles. It is essential

376. Rossolimo, G. I.: (*a*) Pathology of the Spinal Reflexes, Zhur. nevropat. i psikhiat. **2**:239, 1902; (*b*) Le réflexe profond du gros orteil, Rev. neurol. **10**:723, 1902; (*c*) Der Zehenreflex (ein speziell pathologischer Zehenreflex), Neurol. Centralbl. **27**:452, 1908; (*d*) Mein Zehenreflex, Deutsche Ztschr. f. Nervenh. **97**: 172, 1927.

377. W. E. Robertson and H. F. Robertson (Diagnostic Signs, Reflexes and Syndromes Standardized, Philadelphia, F. A. Davis Company, 1939, pp. 238 and 250) were hardly correct in defining the Rossolimo reflex as follows: "When the great toe of the paralyzed side is lightly percussed, or stroked upon its plantar surface, extension or abduction of the toe results. (AUTHORS' NOTE.—This may be regarded as a variant of Babinski's toe sign.)

that the stroke be brief, sharp and elastic, with the hand of the examiner relaxed. The foot must be kept warm.

There is considerable divergence of opinion about the nature of the Rossolimo reflex. Rossolimo himself said nothing about the physiologic nature of his reflex but simply stressed its importance as a pyramidal sign. Goldflam [378] stated that a positive Rossolimo sign proves the presence of an organic lesion of the central nervous system. He stated the opinion that the veil of mystery around the Rossolimo reflex is far from being lifted. After having discussed the Rossolimo reflex in a monograph of 274 pages, Goldflam concluded that it is a pathologic "bone-joint" reflex and is much more closely related to the deep reflexes than to the skin reflexes, especially to the Babinski sign. I do not believe such a vague statement offers anything to the clarification of the issue. Rossolimo, as well as many other neurologists, maintained that his reflex was specifically pathologic and could not be elicited in normal persons. Oddly, most investigators were less concerned with the pathophysiology of the Rossolimo reflex than with its relation to the Babinski reflex. Much confusion has arisen from these discussions, since the reflexes of Babinski and Rossolimo belong to entirely different types.

Lewandowsky [379] listed the Rossolimo reflex under the "bone reflexes." One cannot object strongly enough to such a classification, since it is hardly possible to assume that the stimulation of any bone in any way could in itself affect the plantar muscles and produce flexion of the toes. Bone in itself cannot be regarded as the point of origin of any reflex. It is hardly necessary to dwell on the discussion as to whether or not the Rossolimo reflex is a superficial one. It certainly is not, and one is astounded to find in the popular textbook of Best and Taylor [380] that the Rossolimo sign is listed among "other superficial reflexes"!

To understand the physiology of the Rossolimo reflex, one may best compare it with the finger flexor reflex. The physiologic mechanisms of the two reflexes are identical, so much so that many authors speak of the "finger Rossolimo" and the "toe Rossolimo" sign. In both reflexes, a sudden stretching of the flexors produces their contraction, with subsequent bending of the fingers and toes respectively. The technic is, by the way, the same as that used in elicitation of any other deep muscle reflex, such as the knee or the ankle jerk. Rossolimo [376c] himself mentioned that the contraction of the muscles in his reflex has the same characteristics as that seen in "tendon reflexes."

Both the finger flexor and the Rossolimo reflex are pronounced in cases of pyramidal lesions and may be so exaggerated that clonus of the fingers and of the toes respectively can be elicited in the same manner as the patellar and the ankle clonus. Neither reflex is always seen in normal persons, but both are present in persons with reflexive hyperirritability of any origin.

It must be stressed explicitly that the Rossolimo reflex is in itself not absolutely pathologic. Freund [381] stated: "It is known that occasionally a positive Rossolimo reflex may appear in persons with an intact central nervous system." Kroll [382] asserted that plantar flexion of the toes on tapping of the sole is seen in normal persons. Weingrow [383] observed that the Rossolimo reflex, in part at least, is not rare in normal persons. Bickel [308] observed the Rossolimo reflex in several patients with general functional exaggeration of the reflexes. Kastein [384] succeeded in eliciting the Rossolimo reflex in normal persons by passive stretching of the plantar muscles or by slight voluntary innervation of these muscles. Quensel [385] stated:

> . . . A milder degree of the Rossolimo reflex may be encountered in completely normal persons or in persons who otherwise show some liveliness of the reflexes. This liveliness indicates an insignificant functional nervous hyperirritability.

Krabbe and Ellermann [386] observed the Rossolimo reflex in a case of tetanus in which there was no Babinski sign, but only a general increase of reflexive irritability. Rittmeister [387] found the Rossolimo sign, sometimes bilaterally, in neurotic patients in whom other reflexes were lively or exaggerated. In these patients, some of whom

378. Goldflam, S.: Die diagnostische Bedeutung des Rossolimoschen Reflexes bei Erkrankungen des Zentralnervensystems, Berlin, S. Karger, 1930.

379. Lewandowsky,[80] p. 605.

380. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice, ed. 3, Baltimore, Williams & Wilkins Company, 1943, p. 1473.

381. Freund, B.: Paradoxe Reflexe und ihre Deutung, Dissert., Basel, K. Schahl, 1935.

382. Kroll,[194] p. 193.

383. Weingrow, S. M.: Plantar Tendon Reflexes, Bull. Neurol. Inst. New York **2**:312, 1932.

384. Kastein, G. W.: De aard en het voorkomen van den reflex van het type Troemner, Hoffmann en Rossolimo, Zukowski, Nederl. tijdschr. v. geneesk. **80**: 1187, 1936.

385. Quensel, F.: Ueber Bedeutung und relative Stellung einiger Reflexphänomene, J. f. Psychol. u. Neurol. **40**:205, 1930.

386. Krabbe, K. H., and Ellermann, M.: Recherches sur le réflexe de Rossolimo, Acta psychiat. et neurol. **8**:241, 1933.

387. Rittmeister, J. F.: Die klinische und biologische Bedeutung des Rossolimoischen Reflexes, Schweiz. Arch. f. Neurol. u. Psychiat. **38**:239, 1936; **39**:142, 1937.

were under observation for as long as two years, there was no reason to assume the presence of an early stage of multiple sclerosis. Rittmeister found a Rossolimo sign, also, in normal persons after the feet were exposed to a warm bath. Beck,[150] too, observed a Rossolimo sign in persons with an intact central nervous system. He stressed the fact that a negative response to tests for the Rossolimo sign may be transformed into a positive one when the foot is dorsiflexed. The same maneuver makes a positive Rossolimo sign more distinct. For a long time my associates and I have used this method of dorsiflexion, which is based on the simple fact that every deep muscle reflex can be elicited better, or, if absent, may sometimes be obtained, if the muscle in question has previously been stretched. It is worthy of note that such a keen and experienced observer as Oppenheim [388] once said of the Rossolimo reflex, "I hesitate to attribute to the Rossolimo reflex a decisive importance when it appears as a completely isolated sign." Quensel [389] expressed the same opinion. He asserted that Rossolimo and his pupils overestimated the importance of this reflex. Trömner [389] spoke of the Rossolimo reflex as absolutely unreliable. The nonspecificity of the Rossolimo reflex as a pyramidal sign is documented by the recent work of Finkelstein and Kaczynski,[390] which was based on the examination of 1,000 patients. They assumed that a great variety of centripetal irritations, coming from the internal organs or from different tissues, may occasionally evoke the Rossolimo sign, especially in the constitutionally predisposed person. These authors even attempted to distinguish a pyramidal Rossolimo and a functional Rossolimo reflex.

Rossolimo [391] first described (1902) this reflex as a "deep reflex of the big toe," which could be elicited by tapping the plantar surface of the big toe. Later, he occasionally observed this reflex, also, in normal persons or in persons with functional nervous disease. He then concluded it was better to tap the plantar surface of the other four toes. Flexion of these toes represented, according to Rossolimo, "a special pathologic reflex." It is hard to believe that the action of the big toe and that of the other toes is fundamentally different—that when the plantar surface is tapped, the flexion of the big toe is normal but the flexion of the other toes is pathologic. It is more rational to assume that, owing to mechanical conditions, the elicitation of the flexor reflex of the big toe is easier to achieve than that of the other toes, and thus the latency of the Rossolimo reflex is less pronounced in the big toe than in the four other toes.

Walawski,[392] in his physiologic experiments, observed that the latent period for the Rossolimo reflex, i. e., the time between the tapping of the toes and the reflexive movement, is the same as that for other so-called tendon, bone and periosteal reflexes, and is much less than that of skin reflexes. He concluded that the Rossolimo reflex is not a skin but a bone-tendon reflex. I should prefer, of course, to call it a "muscle stretch reflex."

Keller,[393] in a careful and readable study, obtained electromyographic evidence that on mechanical irritation of the plantar flexors of the toes a deep muscle reflex appears. This response has all the characteristics, especially with regard to reflex time, of the deep reflexes which Keller, as a pupil of Hoffmann, called *Eigenreflexe*.

The only difference between the finger flexor reflex and the Rossolimo reflex is that the latent character of the Rossolimo reflex is more pronounced than that of the finger flexor reflex. One may thus conclude that the Rossolimo reflex, valuable pyramidal sign as it is, is not in itself a pathologic reflex, but represents only a pathologic increase of a normally latent reflex. It is the increase in the tonus of the plantar muscles that brings the Rossolimo reflex to the fore. In this connection, it is remarkable that the Rossolimo reflex is present only when other deep reflexes are exaggerated. Krabbe and Ellermann [386] emphasized that the Rossolimo sign has no specific diagnostic significance for any disease.

The reflex of plantar muscles of the sole, the deep muscle reflex of which represents the Rossolimo reflex, shares the same fate with the reflexes of other muscles of the leg, for instance, the tibialis posterior muscle. It is hardly possible to elicit the tibialis posterior muscle reflex in normal persons, but it can be clearly seen in persons with reflexive hyperirritability of functional or organic cause (Aymes [394]).

388. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, ed. 7, Berlin, S. Karger, 1923, vol. 1, p. 73.

389. Trömner, E.: Alte und neue Extremitätenreflexe, Neurol. Centralbl. **29**:700, 1910.

390. Finkelstein, Z., and Kaczynski, M.: The Sign of Rossolimo in Internal and Surgical Diseases, reviewed, Zentralbl. f. d. ges. Neurol. u. Psychiat. **76**: 599, 1935.

391. Rossolimo, footnote 376 *a* and *b*.

392. Walawski: Research on Rossolimo Reflex by Graphic Method (Polish), reviewed, Zentralbl. f. d. ges. Neurol. u. Psychiat. **66**:728, 1933.

393. Keller: Experimentelle und klinische Ergebnisse der Elektromyographie, in Kraus, F., and Brugsch, T.: Spezielle Pathologie und Therapie innerer Krankheiten, Berlin, Urban & Schwarzenberg, 1932, supp.

394. Aymes, G.: Le réflexe du jambier postérieur, Marseille-méd. **64**:253, 1927.

This concept of the Rossolimo reflex as a normal reflex which exists in latent form, usually not perceptible but becoming apparent at the slightest increase of the tonus of the plantar muscles, explains why the Rossolimo reflex is such an early and reliable sign of a pyramidal lesion and why it may outlast all other pyramidal signs (Müller-Neff [395]). This early appearance of the sign is due to the fact that even the slightest increase of tonus transforms a latent reflex into an apparent one. The reflex is simply a sign of increased muscular tonicity. In accordance with this view is the experience that in cases of fresh cortical hemiplegia the Babinski sign appears immediately, or very soon, and the Rossolimo sign later—not before six weeks—and becomes more pronounced as time goes on and the muscle tonus increases. This characteristic was emphasized by Goldflam [378] and seemed to him to be unexplained. Filimonoff [396] stressed the fact that the reflexes of spinal automatism are more exaggerated in cases with slight increase of tonus, whereas the reflexes of the plantar flexion type, such as the Rossolimo sign, are associated with severe spasm.

Experimental work, too, supports my view that the Rossolimo sign is a muscle stretch reflex, and, as such, is in close relation to the muscle tonus. Kennard and Fulton,[397] in their classic experimental work on chimpanzees, removed cortical area 4 and produced flaccid paralysis of the contralateral extremities, with a Babinski response and decrease of the tendon reflexes. On removal of the premotor area, area 6, they produced spastic paresis of the contralateral extremities, together with forced grasping, an increase in tendon reflexes, the Rossolimo response and fanning of the toes. Thus, in experiments, too, the Rossolimo sign goes hand in hand with spasticity. It is simply a sequel to muscular spasticity. Rossolimo [376c] himself observed that the distinctness of his reflex parallels the degree of tonus of the muscles of the foot.

From all this, one infers that the Rossolimo sign is nothing more than an exaggerated deep muscle reflex of the plantar muscles. It is a muscle-muscle, or a muscle stretch reflex, a reflex of the same kind as that seen in every muscle which can be effectively exposed to brisk stretching. It is, to be sure, in normal persons more latent than many other reflexes. This latent reflex is the basis of the Rossolimo sign. This concept makes it understandable why changes in position of the body or limbs have no effect on the Rossolimo sign (Rittmeister [387]); it can be influenced only by the tonus of the plantar muscles. It explains, also, why the Rossolimo reflex, as well as all other deep muscle reflexes, can best be reenforced by passive stretching of the corresponding muscles, or by their slight, voluntary innervation (Kastein [384]). The latter maneuver, as has been seen, is an efficient method for reenforcement of every deep muscle reflex.

It would be incompatible with this concept of the Rossolimo reflex if the response should appear contralaterally—if tapping of the toes on one side should elicit a reflex also on the opposite side. It is interesting to note that in two studies (Neustadt and Sioli [191] and Taics [398]) devoted to the crossed reflexes, no such phenomenon was mentioned. The entire literature contains only two references to a crossed Rossolimo sign. Goldflam,[378] in his extensive work on the Rossolimo reflex, mentioned only a single case of a crossed Rossolimo sign, and this in a case of a tumor of the right frontal lobe. Rittmeister [387] reported the second case in the literature (case Hub.), associated with multiple sclerosis. In this case the Rossolimo sign was bilateral and crossed. Rittmeister [387] stated: "Until the physiologic basis of the Rossolimo reflex is more clarified, one must be contented simply with registering this phenomenon." What could be the explanation, in the light of the present discussion, for this rare phenomenon of a crossed Rossolimo reflex? In the first place, it is known that the plantar flexors of the toes, which are physiologically extensors, participate, and to a notable degree, in the extensor rigidity of the foot in cases of pyramidal lesions. This activity is sometimes so extreme that the slightest concussion of the foot brings about plantar flexion of the toes. Certainly, every neurologist has seen patients in whom the mere touching of the toes by the examiner's fingers provoked a Rossolimo reflex. In such a patient, the tapping of the toes on one side only, the brisk consecutive bending of the toes and, eventually, the accompanying associated reflexive movements could, through the pelvis, easily transmit the concussion to the opposite side. Such a transmission would be facilitated by the rigidity of both lower extremities, due to the pyramidal lesion. Neither Goldflam nor Rittmeister considered the possibility of such a

395. Müller-Neff, M.: Der Rossolimosche Reflex, Dissert., Leipzig, 1932.

396. Filimonoff, I. N.: Zur Frage über das Wesen der sogenannten pathologischen Reflexe, Ztschr. f. d. ges. Neurol. u. Psychiat. **103**:416, 1926.

397. Kennard, M. A., and Fulton, J. F.: The Localizing Significance of Spasticity, Reflex Grasping, and the Signs of Babinski and Rossolimo, Brain **56**:213, 1933.

398. Taics, I.: Ueber gekreuzte Reflexe, Dissert., Basel, 1936.

transmission in their cases and made no examination for a crossed Rossolimo reflex under conditions which would mechanically prevent such a transmission as much as possible.

The second explanation might be that in the crossed Rossolimo reflex, not the Rossolimo reflex, but the Bernhard-Schrijver phenomenon is involved—a reflex which is not identical with the Rossolimo reflex and can be elicited from a point distant from the foot, since it is not a deep muscle reflex. This phenomenon will be discussed later.

The third, and most probable, explanation of a crossed Rossolimo sign is that the crossed plantar flexion of the toes is a part of the contralateral extensor reflex. This crossed extensor reflex, studied in animals by Freusberg [399] and in man by many observers (the literature has been compiled by Russetzki [134]), consists of an extension thrust of the whole extremity, including flexion of the toes in response to stimulation of the opposite foot—a movement termed by the French *la synergie d'allongement croisé*. The flexion of the toes is only a part, and a very easily induced one, of the extensor reflex, just as the Babinski toe sign is a part of the flexor withdrawal reflex. Thus, by varying the intensity of the stimulus, one might elicit only flexion of the toes on the opposite side. Weak stimuli evoke crossed plantar flexion of the toes, while strong stimuli may evoke contralateral dorsiflexion of the toes with withdrawal of the leg and all other manifestations of the flexor synergy. A weak stimulus is applied for the Rossolimo sign. If the crossed Rossolimo reflex is a part of the contralateral extensor reflex, then it is not a plantar muscle reflex but a response that belongs to a vast group of reflexes of medullary automatism. It is essential to keep these two types of reflexes apart—the muscle stretch reflexes and the reflexes of medullary automatism—and to keep, also, in mind the obvious truth that not every reflexive muscular response to external stimulation is a muscle stretch reflex. With regard to flexion of the toes as a part of the crossed extensor reflex, reference may be made to the work of Parhon and Goldstein,[400] Gordon [401] and Leibowitz.[402]

399. Freusberg, A.: Reflexbewegungen beim Hunde, Arch. f. d. ges. Physiol. **9**:358, 1874.

400. Parhon, C., and Goldstein, M.: Sur le réflexe plantaire contralateral, J. de neurol. et hypnol. **7**:141, 1902.

401. Gordon, A.: Contralateral Plantar Reflex and Its Clinical Interpetation, Tr. Am. Neurol. A., 1920, p. 282.

402. Leibowitz, O.: Ueber einige kontralaterale Wirkungen des Fussohlenreizes, Deutsche Ztschr. f. Nervenh. **95**:123, 1926.

The concept of the Rossolimo sign as a normal but latent reflex which comes distinctly to the fore in the presence of functional or organic reflex hyperirritability, explains best the clinical and experimental facts. Though there is nothing unusual in a reflex being latent or not distinctly perceptible, it must be admitted that the latency of the Rossolimo reflex may be more pronounced than that of any other deep muscle reflex.

The reason for this latency must be sought, first, in mechanical conditions that make it difficult to stretch the muscles directly or to transmit to the plantar muscles the tension of the tendons produced by tapping of the toes. The intricate winding course of these tendons, the normal flexor position of the distal phalanges and the hyperextension of the proximal phalanges may account for this. Mechanical conditions, too, may account for the fact that it is easy to elicit the Rossolimo reflex in normal children from 2 or 3 months to 2 or 3 years of age (Goldflam,[378] Prissmann,[403] Rimbaud and associates,[404] Rittmeister,[387] Partelidès [405] and de Bruin [406]). Here, one must take into consideration the fact that in early childhood the toes are straighter; they become bent later, with the first phalanges hyperextended. This is certainly due in part to the wearing of footgear. In general, it is easier to effect stretching of a muscle if its tendon has a straight course than if it has a bent one. Tapping of the toes transmits more stretching impulse to the plantar muscles when the toes are straight than when they are bent, since in the latter case much of the impulse to the toes is used up to stretch the tendons of the bent toes and does not reach the plantar muscle at all. It is easier to obtain a positive Rossolimo sign in normal persons on tapping the big toe than on tapping any of the others. This is easily explained by the fact that the second to the fifth toe are usually much more bent than the first toe. Accordingly, Rossolimo found that his reflex was much more pathologic when it was present in the second to the fifth toes than when it was elicited on the first toe. The difference in the reaction of the big toe in adults and

403. Prissmann, J.: Der Zehenreflex von Rossolimo bei Kindern in normalen und pathologischen Fällen, Ztschr. f. d. ges. Neurol. u. Psychiat. **77**:259, 1922.

404. Rimbaud, L.; Anselme-Martin, G., and Partelidès, G.: Le signe de Rossolimo: Sa valeur séméiologique, Rev. neurol. **1**:227, 1937.

405. Partelidès, G.: Contribution à l'étude du réflexe plantaire, Thesis, Montpellier, 1937.

406. de Bruin, M.: Ueber das Babinskische Phänomen und über verwandte Reflexe im jüngsten Kindesalter, Acta pædiat. **8**:277, 1929.

that of all the toes in early childhood can thus best be explained on a purely mechanical basis.

We cannot subscribe to the opinions expressed by Astwazaturow,[407] Rittmeister,[387] Borovski[408] and Markow,[409] who saw in the Rossolimo reflex a manifestation of the grasping tendency released by a lesion of the pyramidal tract, or to that of Quensel,[385] who saw in the Rossolimo and in kindred reflexes parts of a greater synergy or of metameric spinal automatism. Neither is it correct to see in the Rossolimo sign, as Grinker did,[410] "a true defense reflex." One can see the grasping tendency in the tonic foot response, in the so-called foot grasping, described first by Schuster and Pinéas[411] in 1926, then analyzed in more detail by Brain and Curran[178] in 1932 and reviewed by Goldstein[412] in 1938. This grasping reflex of the foot, like that of the hand, a reflex which is likewise released through lack of cerebral inhibition, has a characteristically slow and extensive movement. But the Rossolimo reflex is a brisk response to a brisk, passive stretching of the muscles, such as occurs in all deep muscle reflexes. It has no more grasping qualities than does the patellar reflex. The Rossolimo reflex is as far from the grasping reflex of the foot as the finger flexor reflex is from the forced grasping of the hand. These two types of reflexive reaction can be easily distinguished and are in their nature and genesis entirely different.

Smyslow[413] expressed the opinion that the Rossolimo reflex was a support reaction *(Stütz-Reaktion)*. From what is known of the support reaction, as described by Magnus and Schwab, it is hardly necessary to dwell on this assumption, since the Rossolimo reflex has not a single characteristic of the support reaction. The two phenomena are entirely different, both in their mode of elicitation and in their clinical manifestations.

Particular stress is laid here on the fact that the finger flexor reflex and the Rossolimo reflex are in many ways similar. This similarity becomes more striking when one notes that the Rossolimo reflex, like the finger flexor reflex, may be elicited in many, completely analogous, ways. The Rossolimo reflex can be elicited not only by tapping the plantar surfaces of the toes but by tapping the metatarsophalangeal groove. The latter method was preferred by Rimbaud.[414] But the Rossolimo reflex can also be elicited, and sometimes more distinctly, by tapping the ball of the foot. Correspondingly, one may, in exactly the same way, elicit the finger flexor reflex by tapping the heel of the hand.

Markow[409] even had a special name for the reflex he elicited by tapping the ball of the foot. He called it the "distal plantar reflex," but at least he stated that its appearance, even on graphic examination, is the same as that of the Rossolimo reflex.

Flexion of the toes, of the same character as in the Rossolimo reflex, may be achieved when the percussion hammer strikes the planta pedis still farther toward the heel, about the middle of the sole, and this is exactly the method of elicitation of the reflexes described by Zhukovski,[358] in 1910; by Kornilow,[415] about 1910; by von Bechterew,[416] in 1910; by Guillain and Barré,[417] in 1916; by Sicard and Cantaloube,[151] in 1916, and by Weingrow,[418] in 1932! Zhukovski,[358] also spelled Joukovski and Shukowski, described, in 1910, a reflex consisting of flexion of the second to the fifth toes on percussion of the middle of the sole. This reflex was described, also, by Kornilow[415] and has since usually been referred to in the literature as the Zhukovski-Kornilow reflex. This is historically incorrect, since the same reflex was described in 1907-1908 by Yoshimura.[419] He emphasized the reflex character of the movement of the toes on tapping of the sole and proposed the name toe flexion–sole

407. Astwazaturow: On the Biological Nature of the Rossolimo's Symptom, in Neurologie, neuropathologie, psychiatrie. Memoires publiés à l'occasion du jubilé du Prof. G. Rossolimo, 1884-1924, Moscow, Narkomzdrav-Glavnaouka, 1925, p. 300.

408. Borovski, M.: Biogenetic Analysis of the Function of the Footsole, Sovrem. psikhonevrol. **6**:343, 1928.

409. Markow, D. A.: Variants of the Pathological Reflexes, Zhur. nevropat. i psikhiat. **22**:296, 1929.

410. Grinker,[179] p. 140.

411. Schuster, P., and Pinéas, H.: Weitere Beobachtungen über Zwangsgreifen und Nachgreifen und deren Beziehungen zu ähnlichen Bewegungsstörungen, Deutsche Ztschr. f. Nervenh. **91**:16, 1926.

412. Goldstein, K.: The Tonic Foot Response to Stimulation of the Sole, Brain **61**:269, 1938.

413. Smyslow, G. G.: Das digitoperoneale Phänomen und seine Beziehung zu den tiefen Gelenkreflexen, im speziellen zum Phänomen K. Mayers, Deutsche Ztschr. f. Nervenh. **112**:124, 1930.

414. Rimbaud, L.: Réflexes de percussion plantaire, Presse méd. **25**:539, 1917.

415. Kornilow, cited by Markow,[409] Kroll[274] and others.

416. von Bechterew: (*a*) The Tender Point on the Sole and the Sole–Toe Flexion Reflex, Obozr. psikhiat., nevrol. **15**:403, 1910; (*b*) Reflex Phenomenon Associated with Organic Paralysis of the Central Neuron and the New Flexor Reflex of the Dorsum of the Foot, ibid. **16**:385, 1911.

417. Guillain, G., and Barré, J. A.: Le réflexe médio-plantaire, Bull. et mém. Soc. méd. d. hôp. de Paris **40**:516, 1916.

418. Weingrow, S. M.: Plantar Tendon Reflexes, Bull. Neurol. Inst. New York **2**:312, 1932.

419. Yoshimura, K.: Ueber den Mendelschen Fussrückenreflex, in Miura, K.: Neurologia, Tokio, Tohodo-Tanaka, 1909, vol. 6, pt. 3; Wien. klin. Rundschau **22**:309, 1908.

reflex. The literature sometimes mentions the Yoshimura reflex. Von Bechterew,[416] in whose hospital Zhukovski made his observations, confirmed the latter's claims (1910, 1911). He termed this reflex the sole–toe flexion reflex. He could elicit the reflex on tapping the middle of the sole, its outer edge or the heel. He concluded that its nature was identical with that he had previously described as the dorsal foot reflex. But Yoshimura, as well as Zhukovski and von Bechterew, completely overlooked the fact that Sternberg,[8] in his monograph, published in 1893, listed this reflex.

According to Mostvilisker,[420] this response can be elicited from any point on the sole, but best from its posterior part. He as well as Guber-Gritz and Istomin,[365] stated that this reflex was pathognomonic for a lesion of the pyramidal tract and that, next to the Babinski reflex, was the most common of all pathologic reflexes. Zhukovski himself observed that his reflex was present in patients with pyramidal lesions but absent in patients with polyneuritis and tabes and in normal persons. Numerous authors (cited by Markow [421]) reported, however, that they observed this reflex in organically normal persons. Such positive observations are of course more decisive than negative ones. Markow, who made a special study of this reflex, maintained that he had frequently observed a Zhukovski reflex in normal persons and in neurotic patients. He stated:

> . . . It is not allowable to designate the Zhukovski reflex as a pathognomonic sign of a pyramidal lesion, though it is doubtless greatly exaggerated in cases of such a lesion.

Rossolimo,[376d] too, refused to regard the Zhukovski reflex as an essentially pathologic one, since he observed this reflex in 81.7 per cent of normal boys from 9 to 14 years of age. Sternberg [8] listed among the normal reflexes the response elicited on tapping the sole of the foot, which consists of bending of the toes.

In his review of the article by Guber-Gritz and Istomin, Minkowski [422] remarked that the reflex of Zhukovski is apparently closely related to that of Rossolimo. I should go further and say that the two reflexes are identical. They are both muscle stretch reflexes of the plantar flexors of the toes and differ only in that this stretching is effected from different points. This difference is, of course, not an essential factor in consideration of the nature of a reflex. But even these different points may have confluence. As noted before, the Rossolimo reflex can also be elicited by tapping the ball of the foot, and in some textbooks, for instance that of Grinker,[179] this site of elicitation is the only one mentioned. Weingrow [423] even defined the Rossolimo sign as consisting of a plantar flexion of the toes when the ball of the foot is tapped. No less important an investigator than Foerster [424] spoke of the Rossolimo reflex as being elicited by tapping the planta pedis. Rittmeister,[387] in his monograph on the Rossolimo reflex, designated three places for elicitation of this reflex: the ball of all five toes, the plantar surface of the toes and the pulp of the toes. Babonneix [425] stated that the metatarsophalangeal groove was the place of choice. Zhukovski [358] pointed out that his own reflex could be elicited on tapping not only the middle but other points of the sole—its anterior aspect—or the heel. Topographically this anterior aspect of the sole must be regarded as identical with the ball of the foot; thus, the point of elicitation of the Zhukovski and that of the Rossolimo reflex becomes one and the same. One of these reflexes, therefore, cannot be normal and the other pathologic, since it is absurd to suppose that by tapping one and the same spot one can elicit a pathologic as well as a normal muscle stretch reflex. The fact that the Zhukovski sign is observed in normal persons is irrefutable evidence that the Rossolimo sign, too, is in essence a normal reflex. Rossolimo [376c] himself tried to prove that the Zhukovski reflex is not pathologic. He did not realize that by so doing he undermined his own axiom that *his* reflex was a specific pathologic sign. It might be easier, it is true, to elicit the Zhukovski reflex than the Rossolimo reflex in normal persons, but this is not sufficient reason for their being considered different in principle and for one to be regarded as normal and the other as pathologic.

In 1916 Guillain and Barré [417] described a "medioplantar reflex." They stated that, to their knowledge, this reflex had not been mentioned in the various works on symptomatology of nervous diseases. The reflex consisted of flexion of the toes and plantar flexion of the foot when the medial region of the sole was tapped. They found this reflex constant in normal persons,

420. Mostvilisker, A.: The Sole-Toe Reflex, Zhur. nevropat. i psichiat. **18**:51, 1925; reviewed, Zentralbl. f. d. ges. Neurol. u. Psychiat. **44**:310, 1926.

421. Markow, D. A.: Ueber Zehenreflexe vom Plantarbeugetypus, Deutsche Ztschr. f. Nervenh. **112**:281, 1930.

422. Guber-Gritz and Istomin, [365] abstracted by Minkowski: Zentralbl. f. d. ges. Neurol. u. Psychiat. **42**: 639, 1926.

423. Weingrow, S. M.: Tendon Reflexes of the Foot, J. Nerv. & Ment. Dis. **81**:416, 1935.

424. Foerster,[181] p. 267.

425. Babonneix, L.: Le signe de Rossolimo et les réflexes du pied dans les lésions pyramidales, Gaz. d. hôp. **111**:1208, 1938.

exaggerated in patients with lesions of the pyramidal tract and lost in patients with lumbosacral radiculitis, sciatica, polyneuritis and very early tabes. This reflex is, of course, in every way identical with the reflexes of Yoshimura, Zhukovski, Kornilow and von Bechterew! These authors, too, observed, in addition to plantar flexion of the toes, plantar flexion of the foot, a response which is of course merely the achilles reflex. It escaped Guillain and Barré that Sternberg,[8] twenty-three years before, mentioned a reflex which appeared when the medial region of the sole was tapped, with resulting contraction of the gastrocnemius muscle and, less frequently, of the flexors of the toes. This response is identical with the reflex they claimed as theirs.

Sicard and Cantaloube [151] and Rimbaud,[414] although at variance with Guillain and Barré in regard to the nature of the reflex described by the last authors, observed that flexion of the toes on tapping of the sole was constant in normal persons. Without referring to the work formerly done on this subject, Weingrow,[418] in 1932, described as a new discovery this plantar muscle reflex, which he called the "plantar tendon reflex." He stated:

> Another reaction noted in apparently normal persons in whom all the tendon reflexes may be hyperactive is that of plantar flexion of the toes, especially of the outer four, accompanying the plantar flexion of the foot when the region of the tendon of the flexor digitorum longus is tapped.

He could elicit this reflex easily in normal persons. He certainly was incorrect when in his summary he stated that the tendon reflexes of the plantar region described by him had heretofore not been reported in the literature. In a later paper (1935) Weingrow [423] mentioned that he had succeeded in eliciting single reflexes of the abductor hallucis and of the abductor digiti quinti muscle.

The possibility of elicitation of the deep muscle reflex of the plantar muscles by tapping the points of their insertion on the toes, or by tapping the points at the middle of the sole where they traverse the hollow of the arch, reminds one vividly of the similar behavior of the abdominal muscles. The reflex of the latter muscles can be elicited in quite analogous ways: by tapping their points of insertion on the pelvis, or by tapping the middle of the muscle itself as it traverses the abdominal cavity. Since the abdominal muscle reflex can also be elicited, as has been seen, by tapping their points of origin at the costal margin, it is justifiable to assume that the plantar muscle reflex, too, can be elicited at the point of their origin on the calcaneum.

Mostvilisker [420] observed that Zhukovski's reflex could be elicited from any point on the sole, but best from its posterior part. Guber-Gritz and Istomin [365] found that this reflex could be elicited not only by tapping the middle, but by tapping the anterior part, of the sole, as well as the heel. This reminds one that Zhukovski [358] himself, in describing his plantar toe reflex (1910), mentioned that the same flexion of the toes could be achieved by tapping the heel. Von Bechterew,[416] in 1910 and 1911, stressed the fact that flexion of the toes could be achieved also by tapping the outer edge of the sole or the heel. This reflex has since been known as von Bechterew's heel reflex. Markow [409] called it the "proximal plantar reflex."

In 1933, Weingrow [426] summarized his study as follows:

> 1. A new sign for pyramidal tract disease is presented.
>
> 2. It consists of fanning, plantar flexion, or any other movement of the toes following tapping of the heel.
>
> 3. This sign occurs only in definitely advanced involvement of the pyramidal tracts.

Weingrow did not give any physiologic interpretation of the described phenomenon, nor did he correlate it with other flexion reflexes of the toes. In the last edition of his textbook, Bing [117] listed this reflex as a pyramidal sign. Ganfini [427] devoted an article to Weingrow's sign, which in 7 of 74 of his patients alone indicated the presence of a lesion of the pyramidal tract. In the light of what has been said it must be assumed that Weingrow's reflex is neither a new reflex nor a pyramidal sign. It is the same reflex as that described twenty-three years before by von Bechterew and Zhukovski, and is only a modification of the reflexes with various names elicited by tapping the sole. It is simply a plantar muscle reflex elicited from the heel. It might well be a valuable pyramidal sign, but in itself is not pathologic. The same may be said of all reflexes elicited in one way or another through stretching of the plantar muscles.

The mechanism of the reflex elicited on tapping the heel, as described by von Bechterew, Weingrow and others, is easily understood: Owing to the elasticity of the structures of the foot, a blow with a hammer on the plantar surface of the heel will evoke concussion and stretching of the plantar muscles through upward displacement of the calcaneum on which the plantar muscles are inserted. Such a sudden upward displacement of the calcaneum occurs to some degree when one taps the achilles tendon

426. Weingrow, S. M.: The Heel Tap: A Pathological Reflex, J. Nerv. & Ment. Dis. **77**:492, 1933.

427. Ganfini, G.: Su due nuovi segni di lesione delle vie piramidali, Riv. sper. di freniat. **57**:709, 1933.

to elicit the ankle jerk. It is justifiable, therefore, to assume that tapping of the achilles tendon may also provoke bending of the toes. This is indeed so, as pointed out by Bickel [308] in 1912, though it is not often seen. It occurs especially when the reflexes are exaggerated. Yoshimura [419] noted plantar flexion of the toes on elicitation of the achilles reflex in a case of spastic paresis of the legs. Foerster [428] observed that when the reflex of the plantar flexors of the toes is exaggerated, there often occurs a lively flexion of the toes when the achilles tendon is tapped. Rittmeister [387] described in detail a case (Böh) of a complete transverse section of the spinal cord at the level of the twelfth thoracic vertebra in which tapping of the achilles tendon produced simultaneously the achilles and the Rossolimo reflex. Occasionally, plantar flexion of the toes on tapping of the achilles tendon is present when the achilles reflex is lost. Villaret and Faure-Beaulieu,[429] Boveri,[430] Fromentel [431] and Kempner [432] observed reflexive flexion of the toes on tapping the achilles tendon both in cases of disease in which the achilles reflex was lost, such as tabes, and in cases of spastic paralysis and contractures.

In the same way in which a tap on the dorsum of the hand will elicit the finger flexor reflex, a tap on the dorsum of the foot will elicit the plantar muscle reflex. The mechanism is easily understood: Owing to the elasticity of the foot, with its numerous yielding joints, a sharp tap on the dorsum of the foot brings about a brisk depression of the arch and through this, since both ends of the arch are fixed by plantar muscles, a sudden stretching of these muscles. Flexion of the toes by this maneuver was first described by von Bechterew,[433] in 1904, as a tarsophalangeal reflex, or toe flexion reflex, and later, in 1904 and 1906, by Mendel,[434] as a reflex of the back of the foot. It is usually referred to as the Mendel-Bechterew, or the Bechterew-Mendel reflex. With regard to the controversy between von Bechterew and Mendel over the priority for discovery of this reflex, it should be pointed out that Sternberg,[8] in his monograph published many years before, in 1893, listed this response as a reflex elicited from the dorsal aspect of the metatarsals, stimulation of which produces flexion of the toes. The reflex is also called the dorsocuboid, the cuboidodigital and the metatarsal reflex. It is best elicited by tapping the lateral part of the dorsum pedis in its proximal half at a point which corresponds to the base or middle of the third and fourth metacarpal bones, the cuboid bone and the third cuneiform bone.

It is obvious that it is easier to effect a stretching of the plantar muscles by direct tapping of the toes (Rossolimo), by tapping the muscles in the middle of the sole as they pass over the hollow of the arch (Zhukovski) or by tapping the heel (von Bechterew, Weingrow) than indirectly, by tapping and pressing down of the dorsum of the foot (von Bechterew, Mendel). With the last-mentioned technic the solidity of the arch must be overcome; this is considerable, despite its elasticity. This solidity varies, of course, according to the constitution, the age of the person and the condition of the foot.

This purely mechanical difficulty of effecting a flexion of the toes by the maneuver of von Bechterew and Mendel accounts for the fact that this reflex is comparatively infrequent with pyramidal lesions. Osann [435] observed it in 38.6 per cent of cases. Lissmann [436] elicited a Bechterew-Mendel reflex in 6 of 21 patients with hemiplegia. It is easy to understand why the Bechterew-Mendel reflex is much less frequent than the Rossolimo reflex, although, according to many authors, the two reflexes are often elicited together. Rossolimo [376c] himself reported that the reflex of von Bechterew and Mendel was present in 20.8 per cent of 669 cases, whereas the Rossolimo reflex was elicited three and one-half times as often. Feigin,[437] in a series of 160 cases, observed the Zhukovski reflex associated with pyramidal lesions in 56.2 per cent of cases, the Rossolimo reflex in 50 per cent and the Bechterew-Mendel reflex in only 37.2 per cent. Rimbaud and associates [404] noted that the ratio of the frequency of the Bechterew-Mendel reflex to the frequency of the Rossolimo reflex was 19:23. According to Trömner,[438] one can sometimes obtain flexion of the toes more dis-

428. Foerster,[65] vol. 5, p. 149.

429. Villaret, M., and Faure-Beaulieu: La fléxion du gros orteil par percussion du tendon d'Achille, Rev. neurol. **23**:528, 1916; Le signe de la fléxion du gros orteil par percussion du tendon achilléen, Presse méd. **25**:531, 1917.

430. Boveri, P.: Sur un réflexe particulier et pathologique du gros orteil: Le phénomène de la flexion de la deuxième phalange, Rev. neurol. **29**:953, 1916.

431. Fromentel, A.: Thesis, Montpellier, 1916.

432. Kempner, in discussion on Bernhard,[454a] Zentralbl. f. d. ges. Neurol. u. Psychiat. **30**:125, 1922.

433. von Bechterew, W.: Ueber einen besonderen Beugereflex der Zehen, Neurol. Centralbl. **23**:609, 1904.

434. Mendel, K.: Ein Reflex am Fussrücken, Neurol. Centralbl. **23**:197 and 610, 1904; Ueber den Fussrückenreflex, ibid. **25**:293, 1906.

435. Osann, E.: Untersuchungen über den Mendel-Bechterewschen Fussrückenreflex, München. med. Wchnschr. **56**:2468, 1907.

436. Lissmann, P.: Neuere Untersuchungen über den dorsalen Fussrückenreflex, München. med. Wchnschr. **1**:1030, 1907.

437. Feigin, P.: Clinical Examination of the Rossolimo Reflex, Sovrem. psikhonevrol. **3**:294, 1926.

438. Trömner,[389] p. 702.

tinctly by tapping the sole than by tapping the dorsum of the foot. Zhukovski [358] reported cases in which his reflex was positive when the Bechterew-Mendel reflex was negative. When both the Rossolimo and the Bechterew-Mendel reflex are present, the former is usually stronger (de Bruin [400]). In a recent article, Babonneix [425] stated:

> The Mendel reflex is often absent when the Rossolimo reflex is present, and it is never present when the Rossolimo reflex is absent.

In this connection, it is noteworthy that, according to Markow [409] the reflexes of the plantar flexors are absent in the newborn. The Rossolimo reflex appears first and the Bechterew-Mendel reflex later.

According to several authors, such as Acquaderni,[439] Osann,[435] Meyer,[440] Lissmann [436] and Voss,[118] the Bechterew-Mendel reflex is commonly present in infancy. This is best explained by the fact that at this age the arch of the foot is much more elastic than in later life. Morton,[441] in his monograph on the human foot, spoke of the ". . . rigidity of matured tissues that replaces their earlier elasticity." It is quite understandable that the Bechterew-Mendel reflex must be less common with advancing age, as a result of the increased rigidity of the entire structure of the foot. The frequency with which the Bechterew-Mendel reflex is found in patients with multiple sclerosis can be explained in this way, since this disease is most common in relatively young people, in whom the arch of the foot is still very elastic. Unfortunately, there are no statistics on the relation of the Bechterew-Mendel reflex to the age of the patient, nor has any attention been paid to the mechanical and orthopedic conditions of the foot. These conditions, of course, are essential for the appearance or nonappearance of the Bechterew-Mendel reflex. It is easily understandable that the tapping of the dorsum of the foot would more readily produce a depression of the arch, and thus a stretching of the plantar muscles, in a youth with a normal arch and elastic structure of the foot than in a man with a flat foot or in one with diminished elasticity due to sclerotic changes. Little attention has been paid in the literature to the position of the foot on elicitation of the Bechterew-Mendel reflex. Beck [150] observed that in cases of spasticity with talipes equinus the Bechterew-Mendel reflex is absent. This is an important fact, and the reason is easily understandable. In talipes equinus the plantar muscles are shortened and relaxed; of course such muscles cannot be properly subjected to brisk stretching.

Von Bechterew and Mendel asserted that their reflex was a definite sign of a pyramidal lesion. Von Bechterew stressed that his reflex could be elicited only in cases with pathologic conditions. Their pupils Nikitin [129] and Meyer [440] shared this opinion. Rimbaud and Boulet,[442] although realizing the parallel character of the Bechterew-Mendel foot reflex and of the Bechterew carpometacarpal reflex—which in itself is not pathologic—still saw in the Bechterew-Mendel reflex a reliable sign of a pyramidal lesion. In another work, Rimbaud and associates,[404] though aware of the strong relation between the Bechterew-Mendel and the Rossolimo reflex, emphasized the pathologic dignity of the Bechterew-Mendel sign. Other authors, for instance Trömner,[389] Voss [118] and Lissmann,[436] did likewise. Lissmann saw in the Bechterew-Mendel reflex the predecessor of a Babinski sign, the presence of which indicates a pyramidal lesion. Even as late as 1941 the Bechterew-Mendel reflex was regarded as a variant of the Babinski sign (Sejman [442a]). Contrary to these statements, however, there is plenty of evidence that the Bechterew-Mendel reflex cannot in itself be regarded as a definite sign of a pyramidal lesion. Sternberg,[8] years before von Bechterew and Mendel, listed this sign as a normal reflex. Krug [443] found the reflex in 10 per cent of normal persons and in patients with functional nervous disorders. Osann [435] in a series of 50 normal people observed a minimal reflexive movement of the toes in 28, slight abduction in 17 and slight flexion in 11. He emphasized that one needs training and experience to decide whether the flexion of the toes is pathologic or not. Acquaderni [439] elicited the Bechterew-Mendel reflex in normal persons, both in adults and in children; it was present in 50 per cent of the latter. He concluded that no significance can be attributed to the Bechterew-Mendel reflex with regard to a lesion or to incomplete myelination of the pyramidal tracts. Of 646 psychotic patients examined by Bertolini

439. Acquaderni, A.: Il riflesso di Mendel-Bechterew, Riforma med. **25**:407, 1909.

440. Meyer, O. B.: Zur Kenntnis des Fussrückenreflexes, Berl. klin. Wchnschr. **2**:1079, 1907.

441. Morton, D. J.: The Human Foot, New York, Columbia University Press, 1937, p. 182.

442. Rimbaud, L., and Boulet: Réflexe tarso-phalangien, Presse méd. **33**:297, 1925.

442a. Sejman, A. I.: A New Variant of Elicitation of the Pathologic Extensor Reflex of the Big Toe in Affection of the Pyramidal System, Nevropat. i. psikhiat. **10**:101, 1941; abstracted, Zentralbl. f. d. ges. Neurol. u. Psychiat. **103**:593, 1943.

443. Krug, C. W. M.: Ueber den Mendel-Bechterewschen Fussrückenreflex, Dissert., Leipzig, E. Lehmann, 1911.

and Rieti,[444] 25 per cent presented a Bechterew-Mendel reflex, but none a Babinski sign. Tedeschi [445] observed the Bechterew-Mendel reflex in a case of psychogenic disturbance, and Stscherback [446] noted it in cases of functional disorders. Tedeschi [445] stated that it would be wrong to assume the presence of an organic lesion of the central nervous system on the basis of a positive Bechterew-Mendel reflex alone. Markow,[409] who declared that the "Bechterew-Mendel reflex is a pathologic sign," found it in a case of acute muscular rheumatism without any clinical evidence of involvement of the central nervous system. He saw the reflex, also, in a case of myopathy. Oppenheim [388] stated that in general he confirmed the experience of von Bechterew and Mendel with regard to their reflex but advised that when this phenomenon is not pronounced, its significance must be evaluated with caution. Thus, valuable a pyramidal sign as it is, the Bechterew-Mendel reflex cannot in itself be regarded as strictly pathologic.

Neither von Bechterew nor Mendel ever tried to give a physiologic interpretation of their reflex; they were chiefly concerned in establishing its importance, especially its superiority to the Babinski sign. Rimbaud and Boulet,[442] Lewandowsky [357] and others saw in it a bone reflex elicited through percussion of the cuboid bone. Voss [118] made the meticulous distinction between the Rossolimo sign, as a periosteum-bone-tendon reflex, and the Bechterew-Mendel sign, as a periosteum-bone reflex. What applies to the Rossolimo reflex applies as well to the Bechterew-Mendel reflex; no stimulation of a bone or of the periosteum could produce an isolated contraction of the plantar muscles with flexion of the toes. For this is needed a brisk stretching of the muscles involved, which is achieved by a sharp tap on the dorsum of the foot, thus bringing about concussion of the muscles and their stretching through depression of the arch. Whether this reflex appears distinctly, or appears at all, depends not only on the mechanical conditions of the foot but on the tonus of the plantar muscles. Osann [435] stressed the latter point. He brought the appearance of the Bechterew-Mendel reflex into close relation with the hypertonia of the muscles. He stated that such hypertonia, often mild, in the flexor muscles of the toes was the chief prerequisite for the appearance of the Bechterew-Mendel reflex. He stressed the fact that conspicuously often the Bechterew-Mendel reflex is associated with ankle clonus. Lissmann,[436] among others, tried to integrate the Bechterew-Mendel reflex with the Babinski sign. These two phenomena, although they both serve as pyramidal signs, are completely different from the physiologic and the clinical point of view. Suffice it to mention here that the Babinski sign is not dependent on muscle tone but the Bechterew-Mendel reflex is, and to a notable degree.

To Marburg [446a] the Bechterew-Mendel reflex and the Rossolimo reflex seemed identical. He stated that both are not plain deep muscle reflexes, but belong to the group of reflex automatisms of the spinal cord. He called them safety reflexes, or reflexes of attack *(Angriffsreflex)*. The name "safety reflex" is meaningless because all reflexes, of every kind, serve the safety of the animal. From what has been said previously it is evident that the contraction of the plantar muscles on stretching, as it occurs in the Bechterew-Mendel and in the Rossolimo reflex, has as much or as little "attack" quality as has the contraction of the quadriceps muscle when the patellar tendon is tapped.

Foerster [447] stated: "The Mendel-Bechterew reflex is an example of the extension of the reflexogenous zones for the contraction of the plantar flexors of the toes." Much confusion has been caused by the misuse of the popular phrase "extension of the reflexogenous zones." Strictly speaking, there are no reflexogenous zones for elicitation of the deep muscle reflexes, but only for elicitation of the superficial ones. Besides, it is not the extension of the reflexogenous zones that brings the reflex of von Bechterew and Mendel to the fore. The primary, essential point is the increase in tonus of the plantar flexors, which lowers the threshold of their excitability to stretching. This makes it possible to effect a stretching from remote and, so to speak, indirect points.

It has been shown how reflexes bearing different names become confluent on the sole, so that it is impossible to distinguish exactly where the area for elicitation of one reflex begins and that of another ends. This fact of course shows, too, the unity of the reflexes elicited from the sole. The same principle applies to the reflexes elicited from the dorsal surface of the foot. It

444. Bertolini, F., and Rieti, E.: Un réflexe de flexion des orteils étudié chez les aliénés, Rev. neurol. **2**:129, 1923.

445. Tedeschi, E.: Sul riflesso dorso cuboideo del piede, Gazz. d. osp. **28**:1188, 1907.

446. Stscherback, A.: Signe de Babinski et dissociation des réflexes profonds et cutanés. provoqués expérimentalement chez l'homme, Rev. neurol. **16**:408, 1908.

446a. Marburg, O.: Studien über die sogenannten Reflexautomatismen des Rückenmarks, Jahrb. f. Psychiat. u. Neurol. **40**:99, 1920.

447. Foerster,[195] p. 908.

has been found again and again by various authors (cited by Markow [421]) that the reflexes from the dorsum pedis can be elicited from other points than those indicated by the men who first described them. Davidenkow [448] pointed out that Mendel's reflex could be elicited from an area extending dorsally from the point designated by Mendel to the external and internal malleolus. The same reflex may be elicited with greater ease by tapping the foot more laterally and in front of Mendel's point. Nikitin [129] could elicit this reflex also from the area of the outer edge of the base of the fifth metatarsal bone. Yoshimura [419] noted mild flexion of the toes on tapping the intermetatarsal spaces, as did Dumke.[449] Sicard and Cantaloube [151] made the same observation and spoke of a special predorsal reflex, or reflex of the interosseous muscles. Markow [409] designated the cuboid bone and the third cuneiform bone as a place for elicitation of the Bechterew-Mendel reflex, as did Rimbaud and Boulet.[442] Distal to this area he could elicit a plantar flexion of the toes, too, which he called the pseudo Bechterew-Mendel reflex. Although he stated that the two reflexes were morphologically similar and that the zones of the reflexes closely approached each other, he declared them as distinct responses, calling the latter the "distal reflex of the dorsum pedis," or "Markow's reflex." He observed it both in normal persons and in patients with pyramidal lesions. The reflex is best elicited by tapping the base of the third and fourth toes in the interosseous spaces. According to Kroll,[450] this normal reflex (Markow) of the distal dorsal portion of the foot is to be distinguished from the pathologic Bechterew-Mendel reflex elicited from the dorsum of the foot. Such a distinction is neither justifiable nor possible.

Thus, the same plantar flexion of the toes may be elicited more or less easily from many points of the dorsum pedis. The difference is merely quantitative. The areas of elicitation of these reflexes are confluent. There is therefore no justification for creating "new" reflexes and according them proper names. These newly created reflexes differ from the old ones only in that they can be elicited from a point but a few centimeters away from the points of elicitation of the old reflexes.

In this study of the reflexes of the sole, the conclusion has been reached that all of them, despite their slight differences and regardless of the point of their elicitation, from toe to heel, must be regarded as one and the same reflex. The same principle applies to the reflexes of the dorsum pedis. All dorsal foot reflexes work by the same mechanism and achieve the same goal, that is, stretching of the plantar muscles by concussion and depression of the arch of the foot. Furthermore, all the reflexes of the dorsal part of the foot and those of the plantar surface constitute together, for all practical and theoretic purposes, an inseparable unit, since they all involve the same muscles, work through the same mechanism of stretching and differ only in the technical methods which effect the stretching of the muscles. No point on the foot has any monopoly for elicitation of the plantar muscle reflex. This reflex is in itself not easily elicited. The different degrees of participation of the various muscles of the foot do not offer sufficient grounds for differentiation of the reflexes which result from tapping of various points. The reflex often exists in latent form. It can be elicited from one point more easily than from another. It may appear merely on the tapping of a particular spot. Still, all this gives one no right either to regard one of the reflexes as pathologic or to construe them as different varieties of independent reflexes.

One may consider the classic representatives of the reflexes that can be elicited from the planta pedis and the dorsum pedis: the Rossolimo and the Bechterew-Mendel reflexes. Time and again authors have noted and stressed that these two reflexes are "similar." "kindred," "akin," or "closely related" (Voss,[118] Babonneix,[425] Rittmeister [387]). Rittmeister reported that his neurotic patients, with no evidence of organic lesions and with a Rossolimo sign, showed the "related" phenomena of the Bechterew-Mendel, Zhukovski and Kornilow reflexes. It is interesting, in this connection, to note that as far back as 1910, von Bechterew [416a] stressed that the toe flexion reflex, elicited by tapping of the planta pedis, does not "differ essentially" from the reflex elicited by tapping the dorsum pedis. I go a step further and assume that all the reflexes obtained both from the dorsum pedis and from the planta pedis are one and the same (fig. 7). In line with this view are the observations of Yoshimura.[419] In studying the Mendel reflex he noted in spastic conditions plantar flexion of the toes on tapping the plantar or the dorsal surface of the foot or the achilles tendon. He observed that for the elicitation of Mendel's phenomenon the distal parts of the sole were most appropriate. With decreasing excitability it became more difficult to elicit a plantar flexion of the toes from the dorsum pedis, but it was easily elicited from

448. Davidenkow, cited by Markow.[421]

449. Dumke, O.: Ueber den Mendel-Bechterewschen Fussrückenreflex, Dissert., Leipzig, E. Lehmann, 1909.

450. Kroll,[194] p. 194.

the planta pedis. It appeared to him that the reflex of the dorsal surface of the foot is practically a reflex of the plantar surface, and he recommended for the Mendel reflex the name toe flexion–sole reflex. This shows that the two series of reflexes are not only closely related but completely identical.

As it was instructive to compare the toe flexor reflex with the finger flexor reflex, so it is of value to compare the reflexes of the plantar muscles and those of the abdominal muscles (Wartenberg [451]). Both sets of muscles have their superficial, or skin, reflexes. Brain [452] stated, rightly, that the "plantar reflex" is akin to the "abdominal reflex." By the latter he meant what is called here the abdominal skin reflex. Both the plantar and the abdominal muscles have their deep reflexes elicited by stretching of the muscles. In either, one may effect this stretching by tapping either the points of origin or the points of insertion, or by tapping the muscles directly as they traverse a cavity—the abdominal and the plantar cavity, respectively. In both muscles the superficial reflexes are more easily elicited than the deep ones, and the deep muscle reflexes, which are weak or hardly noticeable, become distinct or exaggerated in the case of a pyramidal lesion. The superficial reflexes disappear in both sets of muscles when a pyramidal lesion is present. The only notable difference between the reflexive reaction of the abdominal muscles and that of the plantar muscles is that the latency of the deep muscle reflexes of the plantar muscles is apparently greater than that of the abdominal muscles.

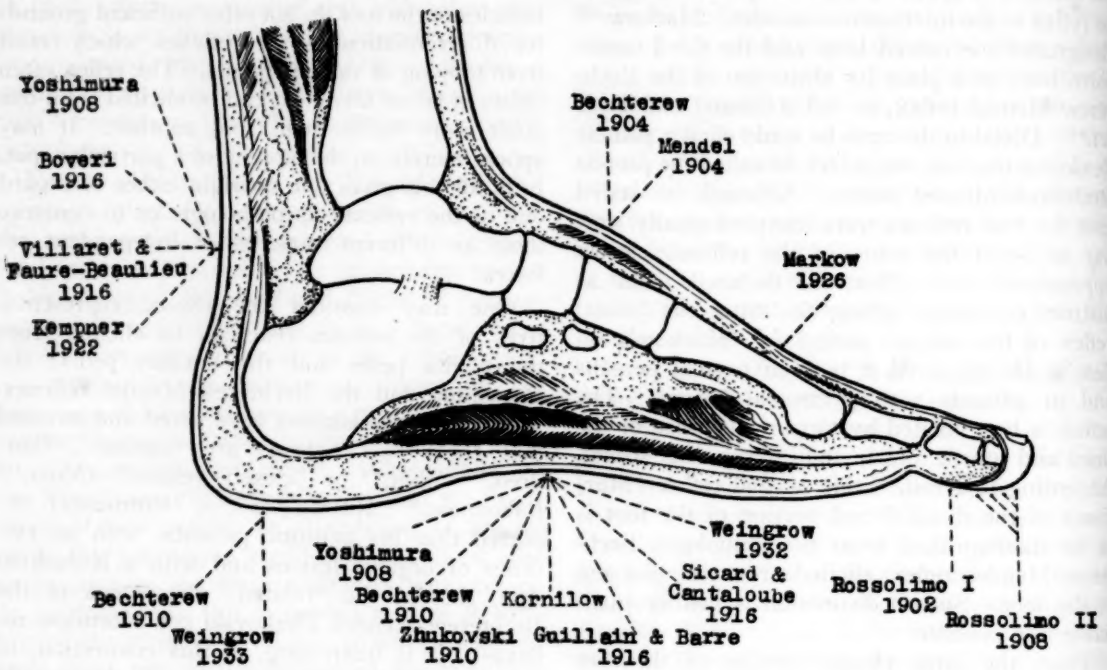

Fig. 7.—Plantar muscle reflex. The points on the surface of the foot indicate the places from which a tap with the reflex hammer can produce a plantar flexion of the toes. The names of the authors who described these reflexes, with the year of publication, are shown. Many of these reflexes are named for their discoverers. All of the eighteen reflexes indicated here represent one and the same reflex: the plantar muscle reflex, as it may be elicited from different points.

DISTANT TOE FLEXOR REFLEX

In 1922, Schrijver [453] and Bernhard [454] independently described a peculiar phenomenon consisting of reflexive plantar flexion of the toes when parts of the body distant from the foot were tapped. This reflex is easily distinguished from the plantar muscle reflex which has just

451. Wartenberg, R.: Reflexes of the Abdominal and of the Plantar Muscles, J. Mt. Sinai Hosp. **9**:832, 1942.

452. Brain, W. R.: Recent Advances in Neurology, ed. 4, Philadelphia, The Blakiston Company, 1940, p. 107.

453. Schrijver, D.: Un réflexe de fléxion des orteils, Rev. neurol. **29**:1425, 1922; Ein Zehenreflex und seine Bedeutung bei Psychosen, Ztschr. f. d. ges. Neurol. u. Psychiat. **79**:398, 1922; Weitere Untersuchungen über den von mir beschriebenen Zehenreflex, ibid. **83**:661, 1923.

454. Bernhard, H.: (*a*) Ueber ein "Zehenreflexphänomen," Berl. Gesellsch. f. Psychiat. u. Nervenkrankh. **6**:26, 1922; (*b*) Ueber ein "Plantarzehenbeugephänomen," Ztschr. f. d. ges. Neurol. u. Psychiat. **80**:589, 1922-1923.

been discussed, and it is essential to make such a distinction. The differentiation can be made on the following grounds: 1. In the distant toe flexor reflex, places so remote from the foot are tapped—for instance, the leg or the thigh—that it is hardly imaginable that the plantar muscles could be mechanically affected in any way. 2. The reflex may occur symmetrically and bilaterally, and this makes the direct implication of the plantar muscles still more improbable. 3. This reflex usually does not occur with pyramidal lesions when the Bechterew-Mendel reflex is positive. 4. This reflex is usually seen when pyramidal lesions are absent, as in cases of psychoses and extrapyramidal diseases (Bertolini and Rieti,[455]). It must be admitted, however, that occasionally the distinction may be difficult. It is a gross mistake, however, to confuse this reflex and the plantar muscle reflex, as Markow [409] and Seletzki [456] did. Unknowingly, the latter discovered for himself the reflex of Schrijver and Bernhard on tapping the outside of the thigh, 6 cm. above the patella. In describing his discovery, he entitled his article, "A New Spot for Elicitation of the Mendel-Bechterew Reflex."

Physiologically, this reflex is by no means a sign of a pyramidal lesion. It cannot be elicited by tapping the foot (Babikow [457]). It is therefore not a Bechterew-Mendel reflex with an enlarged reflexogenous zone (Thiers [458]). It is best classified with the postural reflexes. It might be compared to the "magnet reaction" observed by Magnus in animals. Or it might be regarded as a remnant of a homolateral and contralateral crossed extension reflex. It is apparently tied up with the extrapyramidal system.

It is difficult to give this reflex a name. It is usually called the Schrijver-Bernhard reflex. Aside from the inadvisability of using proper names, such a designation should not be applied, since Marie and Thiers,[459] Lewandowsky [357] and Piotrowski [368] observed the reflex before these authors published their reports. It was rediscovered in 1922 by Markow,[409] in 1925 by Seletzki [456] and in 1927 by de Thurzò.[460] Since the essential point in the technic of elicitation of this reflex is the fact that the plantar muscles are not directly or indirectly tapped or stretched, the name "distant" toe flexor reflex may be appropriate. Clinically, this reflex is insignificant. It does not indicate a pyramidal lesion, is not pathognomonic of any extrapyramidal disease and is, of course, of no value in the diagnosis of mental diseases with which it is allegedly often associated (Bertolini and Rieti [455b]).

In a recent article, Fleischhacker [461] stated that the Schrijver-Bernhard reflex is characteristic for a certain type of subcortical extrapyramidal disorganization, and rightly asserted that this reflex is most likely not due to a lesion of the pyramidal tracts and must not be confounded with the Bechterew-Mendel or the Rossolimo reflex.

Again and again, in my teaching, I have been impressed by the confusion of the student as he struggles with the multitudinous reflexes and their chaotic nomenclature. These studies on reflexes were undertaken primarily with the idea of offering him a simple and comprehensive review of the reflexes in their relation to practical neurologic diagnosis.

This entire study is based on and developed from a few fundamental theoretic postulations on the physiologic nature of the muscle stretch reflexes. Some deductions and practical implications may appear to be too straightforward, too dogmatic and mechanistic, and to simplify unduly the whole complicated matter. But on being confronted with the clinical facts at the bedside, I have always found that what I state here has stood the test of practice. Nothing in my bedside experience thus far has in any way contradicted the deductions set forth in this study. As to the possible reproof for undue simplification of the matter, I may answer that in scientific research, as in artistic endeavor, simplification—the possible reduction to one common denominator—should be the highest aim. Amiel said, in 1861, "The great artist is a simplifier." Nevertheless, some views expressed here seem even to me to be too "radical," and it remains to be seen whether they can stand when confronted by the experience and criticism of others.

I am aware that some of the opinions propounded here contradict those of Babinski, De-

455. Bertolini, F., and Rieti, E.: (*a*) Un riflesso di flessione plantare delle ultime quattro dita studiato nei melati di mente, Rassegna di studi psichiat. **12**:489, 1923; (*b*) Un réflexe de fléxion des orteils étudie chez les aliénés, Rev. neurol. **2**:128, 1923.

456. Seletzki, W. W.: Eine neue Stelle für die Auslösung des Mendel-Bechterewschen Reflexes, Deutsche Ztschr. f. Nervenh. **87**:288, 1925.

457. Babikow, J. K.: Ueber ein Zehenflexionsphänomen bei extrapyramidalen Erkrankungen, Deutsche Ztschr. f. Nervenh. **107**:199, 1929.

458. Thiers, M. J.: Le réflexe de fléxion des petits orteils, Rev. neurol. **1**:720, 1934.

459. Marie, P., and Thiers, J.: Phénomène de fléxion des orteils, Rev. neurol. **20**:599, 1912.

460. de Thurzò, E.: Du réflexe d'adduction des orteils, Rev. neurol. **2**:468, 1927.

461. Fleischhacker, H. H.: Observations on the Toe-Flexor (Schrijver-Bernhard) and Toe-Fanning Reflexes in Catatonic Schizophrenics, J. Ment. Sc. **99**: 403 (July-Oct.) 1943.

jerine, Foerster or Marburg; on the other hand, in studying the literature, I have found that I am supported in my views by similar ones expressed by Sauvage,[462] Tschlenow,[299] Markow,[409] Antoni [137] and others—views that have been neglected or forgotten.

As an instructor, I find the teaching of reflexes along this simplified line highly satisfactory. The concept of reflexes as set forth here appeals to the student, since it is simple, comprehensive and easily understandable.

The history of neurology, and of the reflexes in particular, fills one with modesty and humility and with deep reverence for the past. One is impressed constantly by the wisdom of the old saying, "There is nothing new save that which has been forgotten."

462. Sauvage, R.: Il n'y pas de réflexes osteopériostes, Rev. neurol. **25**:225, 1918.

The history of neurology teaches four lessons:

1. It is difficult to find in the field of neurologic symptomatology anything really new that will stand the test in the light of the research done long before. One should be spared the constant, uncritical rediscovery of time-honored signs, symptoms and reflexes.

2. New neurologic observations should be incorporated in the body of knowledge of the problem as presented in the international literature.

3. The day of Babinski, who, on Feb. 22, 1896, with twenty-eight lines revolutionized the neurologic examination, is no more. The time of purely descriptive symptomatology is over.

4. Modern neurology needs and wants more physiologic orientation. Every new observation in clinical neurology should be subjected to a strict physiologic interpretation.

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INTRAMEDULLARY LESIONS OF THE SPINAL CORD

STUDY OF SIXTY-EIGHT CONSECUTIVE CASES

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SURGICAL TREATMENT

Intensive search through the literature on tumors of the spinal cord reveals a surprisingly incomplete knowledge of the various pathologic types of intramedullary lesions in relation to surgical treatment and postoperative course. It is the purpose of this paper to present as complete an analysis as possible of 68 consecutive cases of intramedullary lesions of the spinal cord in which operation was performed at the University Hospital between 1925 and 1943. In 35 of these cases the lesion was a tumor verified by pathologic diagnosis; in 4 cases tissue for histopathologic study was not obtained, and in 5 cases a hemangiomatous tumor, presenting both extramedullary and intramedullary characteristics, was observed. In 4 cases the clinical diagnosis of intramedullary lesion was without confirmation by operation, and in 20 cases syringomyelia was present.

We have been able to make follow-up observations to date, until death or for periods longer than one year in all but 2 of the 35 cases of tumor with verified pathologic material and in all but 1 case of syringomyelia.

HISTORICAL REVIEW

One hundred and twenty-eight cases are reported in the literature in sufficient detail for analysis. This mass of material is too large to present in summary here, but we have appended a comprehensive bibliography. Certain contributions, however, stand out historically and should be mentioned. Von Eiselsberg, the first surgeon to remove successfully an intramedullary tumor (1907), reported (1913) with Ranzi (1913) total excision of an encapsulated "neurofibrillary sarcoma" from the dorsal region of a woman aged 27. This patient was followed for eight years and was reported as maintaining clinical improvement. In 1911 Elsberg and Beer suggested the two stage surgical technic for treatment of intramedullary tumors. They stated that to incise the posterior columns of the cord vertically over the tumor, allow it to extrude by its own pressure and then to excise this extruded portion later was a safer procedure than to attempt to remove or partially to remove the tumor at a single operation. Since that time little has been added to the operative technic, but a great deal to operative courage. In 1927 Cushing performed a two stage laminectomy, extending from the foramen magnum to the third lumbar vertebra, and totally removed an ependymoma extending from the medulla to the second dorsal segment. Below this level a hydromyelic cavity extended through the remainder of the cord. No report is available as to the progress of this patient after the twenty-five-day postoperative period. Saucier and Trepagnier, in 1935, reported a case of decompression of the entire spinal cord. The cord was not incised, and no specimen was taken for biopsy. The patient received intensive irradiation, and his condition was said to be improved nine months after operation. In 1938 Horrax and Henderson reported a case of a two stage laminectomy extending from the foramen magnum to the third lumbar vertebra. An ependymoma measuring 38.5 cm. in length was removed. The patient's condition was reported as improved fourteen months after operation.

In 1931 Kernohan, Woltman and Adson presented the first histologic classification of intramedullary tumors of the spinal cord. They pointed out that the classification of gliomas of the brain, as suggested by Bailey and Cushing, could be applied to intramedullary tumors of the spinal cord.

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Dr Nathan Malamud aided in the interpretation of the pathologic material, and Dr. Isadore Lampe assisted with the section on roentgen ray therapy.

The surgical treatment of syringomyelia was advanced by the publications of Elsberg (1912) and Puusepp (1926) and by the complete review of Frazier and Rowe (1936).

In addition to the numerous reports of isolated cases and small series of cases of intramedullary tumors surgically treated, the literature contains four numerically important contributions. In 1918 Steinke collected 36 published cases for Frazier's textbook, "Surgery of the Spine." Elsberg (1932) gave detailed information concerning 13 personal cases and, in 1941, made a general statistical analysis of 26 cases. Von Eiselsberg (1932) reviewed his 14 cases, and, similarly, Robineau (1932) reported 15 cases. In all of these series, however, little attempt was made to correlate the results of operation with such important factors as pathologic type of tumor, location, type of operation and radiosensitivity of the growth. It is with the hope of gaining such information that we have attempted to analyze the cases reported in the literature and, in addition, to present our own series of 68 consecutive surgical cases, covering a period of eighteen years at the University of Michigan Hospital.

TABLE 1.—*Incidence of Intramedullary Tumors According to Age (Decades)*

Decade	Literature		Authors' Series	
	Number	Per Cent	Number	Per Cent
0 to 9 years	7	6	3	9
10 to 19 years	18	15	15	14
20 to 29 years	26	20	11	32
30 to 39 years	36	28	4	11
40 to 49 years	22	17	7	20
50 to 59 years	14	11	3	9
Over 60 years	4	3	2	5
	127		35	

TABLE 2.—*Segmental Distribution of Intramedullary Tumors*

Segments	Literature		Authors' Series	
	Number	Per Cent	Number	Per Cent
Whole cord	4	3	1	3
Foramen magnum to dorsal	2	2	2	6
Foramen magnum to cervical	4	3	3	9
Cervical	30	23	3	9
Cervicodorsal	12	9.5	4	11
Dorsal	64	50	10	28
Dorsolumbar	3	2.5	4	11
Lumbosacral	9	7	8	23
	128		35	

TABLE 3.—*Duration of Symptoms of Intramedullary Tumors*

Duration	Literature		Authors' Series	
	Number	Per Cent	Number	Per Cent
Less than 1 year	31	30	15	43
1 to 5 years	55	53.5	9	26
5 to 10 years	6	6	5	14
More than 10 years	11	10.5	6	17
	103		35	

GENERAL ANALYSIS OF INTRAMEDULLARY TUMORS

We have been able to obtain certain information regarding intramedullary lesions treated surgically both from the cases reported in the literature and from our own series. We shall present first the analysis of the cases of intramedullary tumors and shall consider those of syringomyelia in a separate section.

Sex Incidence.—In 2 of the 128 cases reported in the literature, the patient's sex was not mentioned. In 72 (57 per cent) the patients were males and in 54 (43 per cent) females. In our series of 35 verified cases, 18 patients (51 per cent) were males and 17 (49 per cent) females. It is obvious that there is no significant difference in incidence between the sexes.

Age Incidence. — Intramedullary tumors appear most frequently between the ages of 20 and 50. Certainly, they are relatively rare in childhood and advanced age.

Segmental Distribution.—In well over 50 per cent of cases intramedullary tumors of the cord involved the dorsal segments.

Duration of Symptoms.—The relatively long duration of symptoms of intramedullary tumor of the cord is surprising in view of the narrow confines of the cord within the spinal canal and the fragility of the cord itself. Most tumors seem to be slow growing as compared with their prototypes in the brain. Analysis of the 6 cases in our series in which symptoms were of ten years' duration or longer revealed an ependymoma of the high cervical region (case 4), a mixed glioma of the same region (case 28), a dermoid cyst of the upper dorsal region (case 33), an ependymoma of the conus (case 12), a spongioblastoma polare of the conus (case 23) and a dermoid cyst of the conus (case 35). One might suspect that the relatively greater width of the neural canal in the high cervical region and in the conus area favored the long period of tolerance of the tumors.

Roentgenographic Findings.—The roentgenographic examination of the spine was mentioned in the reports of 50 cases in the literature. These roentgenograms were of diagnostic value in only 5 cases (10 per cent). In our series, the findings were of diagnostic value in 6 of 35 cases (17 per cent). This difference may

be due to the increasing knowledge of roentgenology and to the better technical equipment now available. Analysis of the 5 cases in the literature shows that 2 were cases of lipoma of the cervical region; 1 was a case of a so-called intramedullary psammoma of the dorsal region (Feiling, 1920); 1 a case of dermoid cyst of the lumbar region, and 1, a case of ependymoma of the conus medullaris. The lesions in our 6 cases in which characteristic widening of the neural canal was apparent consisted of a dermoid cyst of the dorsal region (case 33) and 5 tumors of the conus: 2 dermoid cysts (cases 34 and 35) 1 spongioblastoma polare (case 23) and 2 ependymomas (cases 2 and 13). It is significant that in our 3 cases of dermoid cyst diagnostic changes were revealed in the roentgenogram and that in 2 of the 3 cases the tumor was of the conus. We have, in addition, reviewed all available roentgenograms and have measured the interpedicular spaces at the known site of the tumor, according to the method of Elsberg and Dyke. In retrospect, then, we found significant widening in 5 additional cases: case 28, of mixed glioma of the high cervical area; case 24, of glioblastoma of the low cervical region; cases 16 and 19, of astrocytoma of the upper cervical region, and case 3, of dorsolumbar ependymoma.

Changes in the Spinal Fluid.—We found 36 cases in the literature in which the Queckenstedt maneuver had been performed. Of these, a partial or complete block was reported in 31 (86 per cent). In our series there was block of the spinal fluid in 25 of the 32 cases (78 per cent), partial in 12 and total in 13 cases. In 2 of our cases no fluid could be obtained on lumbar puncture because of the extension of the intramedullary tumor caudally to fill the lumbar portion of the canal. Xanthochromia of the spinal fluid was found in 31 (61 per cent) of the 51 cases reported in the literature. It was present in 19 (56 per cent) of 34 cases in our series. These percentages agree roughly with those of Elsberg, who noted xanthochromia in 7 (41 per cent) of 17 cases of intramedullary tumor. We can find no significant correlation between the pathologic type of tumor and the presence of spinal fluid block or of xanthochromia.

Myelographic Evidence.—Myelographic studies with the use of poppyseed oil were reported in 33 cases in the literature. The level of the tumor was successfully demonstrated in 28 cases (85 per cent). In all but 1 of our series of 17 cases (94 per cent) the level of the tumor was successfully identified. In the 1 case a discrete intramedullary blood clot was demonstrated at operation. In addition, in 7 cases (44 per cent) the myelograms were sufficiently characteristic to permit a roentgenographic diagnosis of intramedullary tumor. In these cases the contrast medium separated into two fine lateral streams at the level of the tumor, outlining a grossly dilated cord. It should be stated, however, that in only 2 of these cases had the clinical diagnosis been other than that of intramedullary tumor.

Clinical Diagnosis.—A diagnosis of intramedullary tumor of the cord was made in only 25 (34 per cent) of the 74 case reports in the literature in which the preoperative diagnosis was mentioned. In our series the tumor was successfully diagnosed in 11 (31 per cent) of the 35 cases. This low percentage of correct diagnoses is due to the fact that in only 8 cases of our series was there present the classic dissociation of touch and pain and temperature sensation (in only 5 of these 8 cases was the tumor diagnosed as intramedullary). In 4 cases the tumors were correctly diagnosed because of their location in the cervical or cervicodorsal region, with marked involvement of the posterior column and focal atrophy. In 1 case the diagnosis was made because of sacral sparing with otherwise complete transverse myelitis, and in 1 case, on the basis of observations at a previous operation. Certainly, it can be said that the majority of intramedullary lesions present neurologic signs which are due to compression of the cord and are equally common to extramedullary and to extradural tumors, especially those in the dorsal region of the cord.

TABLE 4.—*Type of Operation in a Series of Intramedullary Tumors*

Type of Operation	Literature		Authors' Series	
	Number	Per Cent	Number	Per Cent
Total excision	41	34	7	20
Partial excision	32	26	26	74
Vertical myelotomy	24	20	2	6
Biopsy	13	11	..	
Decompression of cord	3	3	..	
Exploration	7	6	..	
	120		35	

Symptoms.—Analysis of the symptoms in cases reported in the literature and in our series failed to reveal any special syndrome which differentiates intramedullary from extramedullary tumor. Pain was the first symptom in 18 of our cases (50 per cent)—root pain in 12 cases and spinal pain in 6 cases. In 10 cases the first complaint was difficulty in walking; in 1, numbness of the extremities; in 4, weakness of the upper extremities; in 1, bladder difficulties, and in 1, spinal curvature.

Surgical Procedure.—In 34 per cent of the cases reported in the literature and in 20 per cent of our series it was possible to excise the intramedullary tumor completely. Total excision is of course the procedure of choice and should be done whenever possible. The tumors in our cases were all well encapsulated, and either the growth could be lifted out in toto after a vertical incision (myelotomy) had been made over the tumor, or after the capsule was incised an intracapsular removal was made and the capsule then stripped from the surrounding tissue of the cord. Usually, since these tumors lie more posteriorly, this can be accomplished without undue trauma to the cord itself. Fine silk guy sutures, transfixing the tumor, may be used to aid in delicate traction.

In this clinic, our associates and we have not felt it necessary to perform the two stage operation of vertical myelotomy, with later removal of the extruded tumor. Usually, when the vertical incision over the whole extent of the tumor has been made, the tumor will immediately partially extrude itself. After removal of this extruded portion, delicate traction can be used on the sides of the incised cord and further removal of the intramedullary tumor accomplished by gentle manipulation. It should be emphasized that such removal should always stop short of normal-appearing cord tissue. This point may be difficult to determine because of the highly infiltrative character of some of these tumors. In such a case it is much better to attempt only minimal removal of tumor tissue. We feel that the vertical incision chordotomy should extend the length of the tumor.

The 2 cases in which longitudinal incision of the cord and biopsy alone were done were those of a cystic ependymoma with no clear line of encapsulation and of a cystic astrocytoma containing a mural nodule which infiltrated the substance of the cord. We feel certain that mere decompression of the cord by opening the dura alone, without incision of the cord over the tumor, is of little value.

It is not within the scope of this paper to discuss thoroughly the operative technic, but certain refinements seem worthy of note. Information as to the location and type of intramedullary lesion may be obtained by gentle aspiration of the tumor by insertion of a fine hypodermic needle just lateral to the posterior midline. The vertical incision in the cord should be made just lateral to, but as near, the midline as possible. The decision as to which side of the midline is preferable is frequently made possible by the appearance of the tumor in one posterior column of the cord. If the mass is not thus apparent, it is preferable to incise the posterior column on the side representing the more severe clinical signs. It is frequently not necessary to sacrifice the dorsal spinal artery, which can be displaced from the line of incision. Hemostasis usually may be obtained by accurate electrocoagulation of the tiny vessels along the incision, with use of a fine pair of bayonet forceps. If the tumor lies relatively far anterior, the dural attachments of the dentate ligament may be severed, so that the cord lies free in the dural canal. As little as possible of the arachnoid should be disturbed, in order to leave an effective barrier against the invasion of the cord substance by granulation tissue. After total or partial excision of the tumor, the dura should be left widely open for purposes of further decompression. The latter procedure is aided by transverse incisions of the longitudinally cut dura. Observations in cases in which a second operation is performed and the dura has been left open in this fashion have convinced us that the dura reforms with relative rapidity, lining a larger cavity than the original dural sac, so that greater decompression is insured. A tight muscle and fascial closure with fine steel wire is now used in this clinic, and leakage of spinal fluid is rare in cases in which the dura is left open.

TABLE 5.—*General Results of Operation on a Series of Intramedullary Tumors*

Type of Operation	Number of Cases	Improvement	No Change	Condition Worse	Operative Death
Total excision.....	7	4	..	3*	..
Partial excision.....	26	9	5†	8	4
Vertical myelotomy.	2	..	1	..	1
	35	13	6	11	5

* In case 33 there had been notable improvement for five years after the first operation (partial excision of a dermoid cyst). The patient died of ruptured appendix three months after the second operation.

† In cases 18 and 24 no follow-up observations were made after the patient's discharge from the hospital.

General Results.—Of the 28 cases in the literature in which total excision of an intramedullary tumor is reported, improvement was recorded in 26 (93 per cent). In our series, in 4 of 7 cases (57 per cent) in which total excision was done, improvement resulted. The data for these 4 cases follow: In case 2, that of an ependymoma, the patient received adequate roentgen ray therapy and was followed one year and five months; in case 7, that of an ependymoma, the patient received inadequate roentgen ray therapy and was followed one year and eight months; in case 22, that of a spongioblastoma polare, the patient received adequate

roentgen ray therapy and was followed seven months, and in case 32, that of an intramedullary blood clot, the patient was followed six months. The higher percentage of cases (93) reported in the literature in which improvement resulted may be due to the frequency of records of isolated cases and of small series in which only successful results were recorded.

The literature contains 52 cases of partial excision of an intramedullary tumor or of vertical myelotomy over the tumor. Of these, postoperative improvement was reported in 24 per cent. In our series, improvement resulted in 9 of 27 such cases, or in 33 per cent. In only 1 of 13 cases reported in the literature did improvement follow biopsy alone. Our series contains no such case.

Roentgen Ray Therapy.—In table 6 we have arbitrarily divided our 35 cases into three categories in an attempt to evaluate the effects of roentgen ray therapy. In 13 cases so-called moderately intensive irradiation was employed. Because of the varying technics used, one encounters difficulty on attempting to evaluate the radiation dose reaching the tumor site. Our present technic utilizes a single field over the tumor site, a daily dose of 200 r being given until a moderately severe cutaneous reaction is reached. The total dose varies from 2,400 to 3,600 r. The physical factors are as follows: 200 kilovolts; 0.5 mm. of copper plus 1 mm. of aluminum (half-value layer, 0.9 mm. of copper), and a distance of 50 cm. All roentgen ray doses in the appended summary of cases are listed as measured in air, without back scatter. All fields were located posteriorly. Formerly, fields were laid out to each side of the midline, and the central ray was directed toward the spinal cord. The single fields now employed are centrally situated over the spine. In grouping the cases in table 6, we included in the group in which "moderately intense" irradiation was employed all the cases in which the dose approached the amount mentioned. The size of the field varied from 10 by 15 to 12 by 20 cm.

While no definite conclusions can be reached as to the effect of intensive irradiation on intramedullary tumors of the cord, the observation may be of importance that improvement occurred in 4 of 10 cases in which the lesion was partially excised and treated, whereas in only 1 of 9 cases with inadequate or no therapy was there improvement (exclusive of 3 cases of dermoid cyst and 1 of intramedullary blood clot). This observation is mentioned only as of possible significance, since such factors as the type and size of the tumor and the type of operation must necessarily play a large role in the ultimate result of roentgen ray therapy. The 4 cases of partial excision in which sustained improvement followed intensive irradiation were: case 5, that of an ependymoma followed six years; case 12, that of an ependymoma followed 6½ years; case 11, that of an ependymoma followed six months, and case 28, that of a mixed glioma followed three years and three months. It is, perhaps, of added significance that ependymomas appear to respond most favorably to roentgen ray therapy.

TABLE 6.—*Results of Roentgenographic Therapy of Intramedullary Tumors*

Type of Roentgen Ray Therapy	Total Number of Cases	Total Excision (7 Cases)			Partial Excision (28 Cases)			
		Improvement	No Change	Condition Worse	Improvement	No Change	Condition Worse	Operative Death
Moderately intense	13	2	..	1	4	1	5	..
Inadequate	9	1	..	..	2	4	2	..
None	13	1	..	2	3	1	1	5
	35	4	0	3	9	6	8	5

The following case is presented in summary because we feel it to be of unique interest and importance, since at a second operation, two years after partial excision of an extensive intramedullary tumor, it was possible to observe directly the effect of operation and intensive roentgen ray therapy.

CASE 30.—W. P., a man aged 23, over a period of two years had noted slowly progressive spastic weakness of both legs, atrophy and ataxia of the left hand and retention of urine. Neurologic examination revealed loss of vibratory sensation and sense of motion and position in the left hand and in both lower extremities. Perception of pinprick was diminished on the left side and absent on the right side below the sixth cervical dermatome. Touch and temperature senses were not involved. The small muscles of the left hand were atrophic. There was spastic paresis of both legs, more marked on the left side, with bilateral extensor plantar responses. No pain or tenderness was noted. Spinal puncture revealed complete manometric block, no xanthochromia and a 4 plus reaction for globulin. A myelogram taken after injection of poppyseed oil revealed a complete block at the sixth dorsal segment, not suggestive of an intramedullary lesion.

In 1941 laminectomy from the fourth cervical to the second dorsal vertebra, performed by Dr. E. A. Kahn, revealed a large intramedullary mass in this region.

Vertical myelotomy was done the entire length of the tumor, the left posterior column being incised. There was immediate extrusion of soft, grayish, gelatinous tumor tissue. The extruded portion was excised and the dura left open for purposes of decompression. The patient was given intensive roentgen treatment one week after operation. An area measuring 36 by 10 cm. treated as two fields, of 10 by 15 cm. each, received 200 r daily over each field, for a total of 3,200 r. The patient improved rapidly and recovered to a point at which his condition was about the same as before operation. It then remained stationary for one and a half years. He was able to walk a mile (1.6 kilometers) and to carry on full time work as a clerk. His condition then began to regress. His legs and the left hand became weak, his gait unsteady and his bladder control poor, and he was forced to stop work. He reentered the hospital two years after operation, with the same neurologic signs as on his previous admission, but of greater severity.

In 1943 a laminectomy from the foramen magnum to the previous operative site, as well as of the second and third dorsal vertebrae, was performed by one of us (W. W. W.). The dura had formed a larger sac and showed no adhesion to the cord. Incision of the dura revealed no gross sign of tumor over the previously incised area of the cord. The posterior columns lay separated and appeared grayish and atrophied. No tumor lay in this exposed trough. Inferiorly, however, a nubbin of grayish tumor tissue protruded through the lowermost tip of the previous incision. This was partially excised. At the cephalad limit of the old incision the cord bulged up to the foramen magnum, and tumor tissue literally gushed forth on vertical incision. Again, it was noted that the tumor tissue existed only within the portion of the cord which had not been incised previously. The myelotomy incision on the left was carried up into the dorsal surface of the inferior cerebellar peduncle. The tumor showed no signs of diminishing in size at this level. It was thought injudicious to incise the medulla higher, and so all extruded tumor tissue was excised and a large part of the remaining tumor gently sucked away from within the cord. The dura was left open and was cut transversely. The muscles and fascia were closed with steel wire sutures, the subcutaneous tissues with chromic surgical gut and the skin with black silk. The patient, under anesthesia induced with tribromoethanol, had shown only a few moments of respiratory irregularity at the time the cervical chordotomy had been done. His convalescence was not remarkable, and he is at present in the hospital receiving roentgen irradiation over the high cervical and suboccipital regions. His left arm is weak and notably ataxic, and he has moderately increased spastic weakness of both legs, although he is beginning to walk with aid. Sensory examination reveals loss of vibratory and position senses below the sixth dorsal segment on the right side and below the clavicle on the left side. Pain and temperature sensations are absent on the right side below the third dorsal segment, with diminution in the saddle area; on the left it is absent below the trigeminal-cervical border to approximately the eighth dorsal segment; inferior to this level it gradually increases until the saddle area is reached, where it is normal. Touch sense is diminished over the aforedescribed area on the right side and is absent on the left side from the third cervical to the first lumbar dermatome, increasing below this to the saddle area, where it is normal. The patient can identify toes on the left side, but not on the right. He has urinary retention, with apparently slightly diminished desire for micturition, and constipation, with some weakness of the anal sphincter.

Pathologic examination of specimens removed at each operation revealed no change in the cellular character of the tumor. Owing to the extremely edematous nature of the growth, a definite diagnosis cannot be made. There was a mucin-like matrix. The cells were distorted, with no discernible boundaries, in places suggesting deformed astrocytes. The diagnosis was glioma, of undetermined type.

The absence of tumor tissue in the area of the former vertical chordotomy and its presence at either end of the incision, although these areas were well within the field of intensive roentgen ray therapy, suggest that after release from its intramedullary position the tumor became susceptible to the roentgen radiation, possibly on the basis of lack of nutrition and unfavorable environment. This observation offers further recommendation for vertical incision over the entire length of an intramedullary tumor of the cord, even though only partial excision can be accomplished.

Mortality.—There were 5 operative deaths in our series, a mortality rate of 14 per cent. In case 10, that of an ependymoma of the whole cord, the patient died twenty-four hours after the second operation, of respiratory failure, convulsions and high fever. In case 13, that of a diffuse fibrillary astrocytoma in the dorsal region, the patient died immediately after operation, of respiratory failure, believed by the pathologist to be due to a persistent thymus—a so-called thymic death. In case 16, that of a fibrillary astrocytoma of the high cervical region, gradual onset of complete transverse myelitis developed twenty-four hours after operation, and death occurred from respiratory failure. In case 23, that of a recurrent spongioblastoma polare of the conus and cauda equina, the patient died of shock on the operating table. In case 15, in which a fibrillary astrocytoma was removed from the foramen magnum to the sixth dorsal segment, and was observed at autopsy to extend into the medulla as high as the olives and downward as far as the low lumbar segments, the patient died eighteen days after operation, after a week of severe, uncontrollable vomiting. There is no doubt that a diffuse intramedullary tumor of the cervical region offers a much greater operative risk than a tumor located in lower segments, owing to the danger of diaphragmatic paralysis.

In addition to these 5 operative deaths, we have information on subsequent deaths in 6 cases. In 3 of these, the cause of death was probably unrelated to the intramedullary tumor: In 1 case death was due to a cerebrovascular

accident; in 1, to pneumonia, and in 1, to an undiagnosed ruptured appendix (because of loss of sensation and masking of diagnostic signs). In the remaining 3 cases the postoperative course was gradually downhill: A patient with cervicodorsal glioblastoma multiforme died two months after operation; a patient with dorsolumbar oligodendroglioma, in four and a half months, and a patient with an intramedullary melanoblastoma, in six months.

Postoperative Follow-Up Observation.—One of the difficulties in evaluation of the results of operation in patients with intramedullary lesions has been the relatively short period of postoperative follow-up observation. Thus, in the literature the average period of such observation was one year and eleven months. We have been fortunate in having an average period of two years and eight months. In table 7 we present the length of survival of patients according to our last information regarding them.

TABLE 7.—*Survival in Cases of Intramedullary Tumors*

Type of Tumor	Total	Operative Deaths	Year of Last Check-Up: 0-1	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	Survival Over 10 Yr.	Longest Period of Survival
Ependymoma	12	1	1*	3	1	..	1	1	1	1	..	..	2	12 yr.
Astrocytoma	7	3	1†	1	1	..	..	..	1	..	..	..	..	6½ yr.
Spongioblastoma polare	4	1	3	..	..	..	..	..	..	..	..	..	..	8 mo.
Glioblastoma multiforme	2	..	2†	..	..	..	..	..	..	..	..	..	..	2 mo.
Oligodendroglioma	1	..	1*	..	..	..	..	..	..	..	..	..	..	4½ mo.
Mixed glioma	2	..	..	..	1*	1	..	..	..	..	..	..	..	3 yr.
Unclassifiable	2	..	..	2	1	..	..	..	..	..	..	..	..	21 yr.
Melanoblastoma	1	..	1*	..	..	..	..	..	..	..	..	..	..	6 mo.
Intramedullary clot	1	..	1	..	..	..	..	..	..	..	..	..	..	6 mo.
Dermoid cyst	3	..	..	..	1	1	..	..	1*	..	..	..	..	6½ yr.
	35	5	10	5	5	2	1	1	3	1	..	..	2	
Syringomyelia	20	1	5 (4*)	4 (1*)	2	1	1	2 (1*)	..	2	..	1	1	13 yr.

* Patient known to be dead.
† No follow-up observation after patient's discharge from the hospital.

TABLE 8.—*General Clinical Data in Cases of Intramedullary Lesions Classified According to Pathologic Type*

Type of Lesion	Total No. of Cases	Sex: Male	Sex: Female	Age Groups, Yr.: 0-9	10-19	20-29	30-39	40-49	50-59	60+	Segments*: Entire Cord	FM-D	FM-C	C	C-D	D	D-L	L-S	Duration of Symptoms, Yr.: 0-1	1-5	5-10	10+
Ependymoma	12	6	6	1	1	3	3	3	1	..	1	..	..	1	2	4	1	3	4	3	3	2
Astrocytoma	7	5	2	..	3	2	..	..	2	..	..	1	2	1	..	2	1	..	3	2	2	..
Spongioblastoma	4	2	2	..	..	2	..	2	..	..	..	..	..	..	..	1	1	2	3	..	..	1
Glioblastoma	2	1	1	1	..	1	..	..	..	..	..	..	..	1	1	..	..	..	2	..	..	..
Oligodendroglioma	1	..	1	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	1	..	..	..
Mixed glioma	2	1	1	..	..	..	1	..	..	1	..	..	1	..	..	1	..	..	1	..	..	1
Unclassifiable	2	1	1	..	..	2	..	..	..	..	..	1	..	..	..	1	..	..	1	1	..	..
Melanoblastoma	1	..	1	..	..	..	..	..	..	1	..	..	..	..	..	..	1	..	..	1	..	..
Blood clot	1	1	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..
Dermoid cyst	3	1	2	1	1	..	..	1	..	..	..	..	..	..	1	..	..	2	..	1	..	2
	35	18	17	3	5	11	4	7	3	2	1	2	3	3	4	10	4	9	15	9	5	6
Syringomyelia	20	11	9	..	2	7	2	7	2	..	..	..	3	4	11	..	..	2	3	9	2	6

* FM indicates the foramen magnum; C, the cervical portion of the cord; D, the dorsal portion; L, the lumbar portion, and S, the sacral portion.

PATHOLOGIC TYPES[1]

GLIOMAS

We have found, in reexamining the 30 gliomas in our series, that the classification as presented by Kernohan, and adapted by him from the original classification of gliomas of the brain by Bailey and Cushing, is simple and inclusive. We have little to add to his original pathologic work but have been able to verify most of his statements. Thus, gliomas of the spinal cord may be divided into the same types as those of the brain. Many, however, present a mixed cell type and varying degrees of cellular differentiation. We have classified these tumors according to the predominant cell type and have utilized Kernohan's subgrouping of the ependymomas into type I (epithelial), type II (cellular) and type III (myxopapillary). The term glioblastoma multiforme is used synonymously with spongioblastoma multiforme. Our series contains no example of a primary intramedullary medulloblastoma, a ganglioneuroma or an astroblastoma. Vascular tumors, such as angioma and hemangioblastoma, are not included because of their extramedullary characteristics or because there was no pathologic material available in

1. The pathologic diagnosis of these tumors was confirmed by Dr. Nathan Malamud, of the neuropathologic laboratory of the Neuropsychiatric Institute.

the cases in which the tumor appeared purely intramedullary. We have experienced diagnostic difficulty in reviewing the pathologic material, owing to lack of original material, scarcity of specially stained sections from the older specimens and cellular distortion due to trauma to the frequently small bits of tumor removed at operation. In table 10, we have compared our pathologic material with that of Elsberg (1941) and that of Kernohan, using the latter's publication of 1932, rather than the more recent work of 1941, since it includes tumors of the filum terminale.

Ependymoma (12 cases).—The pathologic characteristics of intramedullary ependymoma have been thoroughly reviewed by Kernohan, as well as by Wolf, in Elsberg's text. We shall be concerned chiefly with clinical observations. Of the 12 cases of ependymoma in our series, 6 were of males and 6 of females. The youngest patient was a 6 year old girl, with a cervicodorsal tumor (case 5). The oldest patient was a woman aged 51, with a cervical lesion (case 4). The average age was 31 years. In 1 case the tumor involved the cervical segments alone; in 7 cases, the dorsal segments; in 3 cases, the lumbosacral region, and in 1 case, the whole cord. The shortest period of preoperative symptoms was one month (case 9). In this case there was a sudden onset of pain in the lower part of the back and of bilateral sciatic pain. The longest duration of symptoms was that in case 4—a twelve year period of gradually increasing weakness of the right arm. The average duration of symptoms was four years and two months. In only 2 cases did roentgenograms show widening of the neural canal, the tumor in both these cases lying in the conus medullaris. No significant difference from other intramedullary tumors was revealed in the spinal fluid, either by myelographic or by clinical examination.

In 3 cases the ependymoma was well encapsulated and could be removed in its entirety. One tumor extended from the fourth cervical to the sixth dorsal segment; another, from within the conus down to the sacral canal, and 1 was a small lesion limited to the second dorsal segment. The remaining 9 tumors were all infiltrating. The degree of infiltration varied greatly, most of the tumors, however, having areas which were well encapsulated. There was 1 operative death (case 10), following a second operation for a tumor involving the whole cord.

In 2 of the 3 cases in which the tumor was totally excised the condition improved; that in the third became worse. Of the 4 cases in which partial excision plus moderately intense roentgen ray therapy was employed, improvement occurred in 3 and no change in 1, whereas of the 4 cases in which partial excision plus inadequate or no roentgen ray therapy was given, improve-

TABLE 9.—*Results of Operation for Intramedullary Lesions According to Pathologic Type*

Type of Lesion	Total No. of Cases	Type of Operation			Roentgen Ray Therapy			Result of Operation			
		Total Excision	Partial Excision	Vertical Chordotomy	Moderate Intensity	Inadequate	None	Improvement	No Change	Condition Worse	Operative Death
Ependymoma	12	3	8	1	6	4	2	6	3	2	1
Astrocytoma	7	..	6	1	..	3	4	2	1	1	3
Spongioblastoma polare	4	1	3	1	3	..	1	1	..	2	1
Glioblastoma multiforme	2	..	2	..	..	1	1	..	1	1	..
Oligodendroglioma	1	..	1	..	1	..	..	..	..	1	..
Mixed glioma	2	..	2	..	1	1	..	1	1	..	..
Unclassifiable	2	1	1	..	1	..	1	..	..	2	..
Melanoblastoma	1	..	1	..	1	..	..	..	..	1	..
Blood clot	1	1	..	..	..	..	1	1	..	..	..
Dermoid cyst	3	1	2	..	..	..	3	2	..	1*	..
	35	7	26	2	13	9	13	13	6	11	5
Syringomyelia	20	..	..	20	3	3	14	2 (1†)	2	15	1

* The patient was greatly improved for five years after the first operation (partial excision of a dermoid cyst).
† The patient's condition was unchanged for five years and then improved for seven years, with a downhill course for the past year.

TABLE 10.—*Comparison of Three Series of Cases of Intramedullary Glioma*

Type of Glioma	Kernohan's Series	Authors' Series	Elsberg's Series
Ependymoma			
Type I (epithelial)	6	3	
Type II (cellular)	8	7	
Type III (myxopapillary)	7	2	
	21 (47%)	12 (40%)	16 (73%*)
Astrocytoma	4 (9%)	7 (23%)	5 (23%)
Astroblastoma	2 (4%)	..	..
Spongioblastoma polare	2 (4%)	4 (13%)	..
Glioblastoma multiforme	5 (11%)	2 (7%)	1 (4%)
Oligodendroglioma	2 (4%)	1 (3%)	..
Mixed glioma	..	2 (7%)	..
Unclassifiable	3 (7%)	2 (7%)	..
Medulloblastoma	4 (9%)	..	..
Ganglioneuroma	2 (4%)	..	..
	45 (100%)	30 (100%)	22 (100%)

* Since we have not included tumors of the filum terminale or the cauda equina, we have not listed Elsberg's 4 cases of extramedullary ependymoma of the lumbar sac.

ment resulted in only 1. In 2 cases the condition was unchanged, and in 1 it was worse. From this small number no definite conclusions can be reached, but it is perhaps of significance that more improvement seemed to occur with adequate roentgen ray treatment than without it. Case 10, that of a 6 year old girl, illustrates this point. The patient now returns to the clinic, six years after partial excision and adequate roentgen ray therapy, with no complaints or neurologic signs. Before operation she had atrophy and sensory loss over the fifth and sixth dermatomes, absence of the biceps and triceps reflexes bilaterally, absence of the abdominal reflexes, nocturnal enuresis and tenderness on motion of and pressure over the cervical portion of the spine.

When the pathologic material was divided into the three subgroups of Kernohan, it was noted that the 3 tumors of type I (epithelial) all contained some areas characteristic of type II. Two of these tumors had cystic areas, and 1 was solid. Of the tumors falling in type II (cellular), 1 was cystic; 2 were associated with cystic dilation of the cord beyond the limits of the tumor, and 4 were solid. All tumors of type III (myxopapillary) were solid. No significant clinical features or results following operation could be assigned to any particular cellular type.

One patient (case 11) was followed for thirteen years and six months. The shortest follow-up period was that in case 6, in which death occurred six months after operation, from a cerebrovascular accident. Perhaps the most outstanding of our series of cases of ependymoma was case 12, that of a girl aged 16, who was operated on by Dr. Max Peet six years and six months prior to the time of this report. Before operation she had flaccid weakness of both legs, walked with great difficulty and had urinary incontinence and notable diminution in all forms of sensation below the third lumbar dermatome. Partial excision of an infiltrating ependymoma of the conus was performed, and the patient was given moderately intense roentgen ray therapy. She is now walking almost normally; her sensory loss has greatly diminished and her bladder control is normal. She has had 2 normal children and does all of her own housework.

Astrocytoma (7 cases).—Five patients were males and 2 females. The youngest patient was a 10 year old girl, with a tumor of the dorsal region. The oldest was a man aged 55 with a tumor of the upper cervical region, extending through the foramen magnum. In 3 cases the tumor involved the cervical segments alone, and in 4, the dorsal segments; in no case did it involve the lumbosacral segments alone. The shortest period of symptoms was two and a half months, with severe pain around the lower margins of the ribs. The longest period was nine years, beginning with torticollis and dorsal kyphoscoliosis. The average duration of symptoms was three years and two months. There was nothing in the spinal fluid, the roentgenograms, the myelograms or the clinical examinations which was characteristic of astrocytoma alone.

In no case could the astrocytoma be totally excised, owing to the infiltrative character of the tumor. The longest lesion extended from the foramen magnum to the fifth dorsal segment.

The results of operation were poor. In 3 cases the patient died at operation; in 2 cases only slight improvement occurred; in 1 case the condition was unchanged, and in 1 case it was worse. In none of the cases was roentgen ray therapy adequate. The longest period of follow-up observation is now six years and five months; the patient's condition is only slightly improved, but no signs of progression are apparent.

All the tumors were of the fibrillary type; none was of the protoplasmic type. Three contained cystic cavities. One was accompanied by a cystic intramedullary dilatation of the cord. Three were solid tumors.

Spongioblastoma Polare (4 cases).—Two patients were males and 2 females. The youngest patient was a man aged 22, with a well encapsulated tumor of the dorsal region; the oldest was a woman aged 48, who had had partial excision of an intramedullary tumor of the conus fourteen years previously. She had had almost complete relief for seven years; then flaccid weakness of both legs gradually developed and progressed to flaccid paraplegia. She was operated on a second time and died of shock on the operating table.

The tumors were distributed throughout the cord, occurring in the cervical, the dorsal and the dorsolumbar region and in the conus and the cauda equina. The longest tumor extended from the ninth dorsal to the second lumbar segment of the cord.

The shortest duration of symptoms was four months, and the longest, in the case of the recurrent tumor previously mentioned, fourteen years. In the other 2 cases symptoms were present only five and eight months respectively.

The spongioblastomas presented no significant differences in the spinal fluid, the roentgenogram, the myelogram or the clinical findings.

Only in case 22 was the tumor well encapsulated. In this case, that of a man aged 22, complete transverse myelitis had rapidly de-

veloped, over a period of four months, at the level of the tenth dorsal segment except for slight sacral sparing of all forms of superficial sensation. At operation, seven months ago, one of us (W. W. W.) removed a well encapsulated, solid tumor from the dorsal intramedullary portion of the cord, the mass extending from the tenth to the twelfth segment. The patient received a course of moderately intense roentgen ray therapy. His condition is now greatly improved and continues to improve. He walks without assistance, although his gait is spastic-ataxic. Sensory loss is limited merely to diminution of all superficial forms. Vibration and position sensations are lost in both lower extremities. Surprisingly, he has no bowel or bladder difficulties, although before operation he had no control. He is working full time in a factory. In the other 3 cases an infiltrating tumor was partially excised and the 2 surviving patients were given adequate roentgen ray therapy. Their status has not improved. In 1 case the characteristic signs of Recklinghausen's neurofibromatosis were present. Histologically it was difficult to differentiate between spongioblastoma polare and fibrillary astrocytoma, the cell type of the former being slightly more predominant.

Glioblastoma Multiforme (2 cases).—One patient was a man aged 27, and the other, a girl aged 7 years. The tumors extended from the fifth to the eighth cervical and from the seventh cervical to the seventh dorsal segment respectively. The duration of symptoms was short—three and two months respectively. Both patients were tetraparetic and were in poor condition at the time of operation. In both cases the tumor was infiltrating, was moderately soft and was partially excised. In neither case was adequate roentgen ray therapy given after operation. In the first case the condition was unchanged after two months in the hospital, and the patient has not been heard from since. The girl grew progressively worse and died within two months. The histologic picture of both tumors was typical of glioblastoma. It is interesting to note the age (7 years) of 1 patient with this tumor, which in the brain usually occurs in older persons.

Oligodendroglioma (1 case).—A woman aged 43 had an infiltrating intramedullary tumor of the conus, which extended downward into the lumbar sac. Her symptoms, starting with pain low in the back and over the distribution of the sciatic nerve bilaterally, progressed over a four week period to complete flaccid paralysis of both legs, with diminished sensation below the first lumbar segment. The tumor was partially excised, and the patient was given moderately intense roentgen ray therapy. Her condition continued to grow worse, and she died four and a half months after operation. The tumor histologically, unlike most oligodendrogliomas of the brain, contained no calcareous deposits and showed relatively malignant cellular characteristics.

Mixed Gliomas (2 cases).—The patients were a man aged 37 and a woman aged 68. Histologically, both the tumors consisted of elongated cells with distinct processes arranged in perivascular pseudorosettes. Both contained scattered giant cells. The cellular characteristics suggested those of both ependymoma and spongioblastoma polare. One tumor was from the upper cervical area and the region of the foramen magnum, and 1, from the upper dorsal region. In 1 case the symptoms were of seven months' duration, and in 1 they had been present ten years. The clinical findings were not different from those in cases of other intramedullary tumors in our series.

Both tumors were solid and showed necrosis in areas, and they were partially excised. One patient was given adequate roentgen ray therapy and has gradually improved over a period of three years and three months. The other patient received inadequate roentgen ray therapy; the condition remained unchanged, except for relief of her original root pain, and she died two years and three months after operation.

Unclassifiable Tumors (2 cases).—One of the cases of these tumors (case 30) has been summarized in the section on roentgen ray therapy; in the other (case 29) there was a history of rapid development of a partial Brown-Séquard syndrome for two months. The tumor, which was totally excised, extended from the fifth to the eighth dorsal segment and was well encapsulated and firm. Histologically, it consisted of large cells, frequently of foam type, with lipid content. There were many areas of lymphocytic elements. No proliferation of connective tissue, either of the collagen or of the reticulin type, was evident. The patient was given moderately intense roentgen ray therapy. She has been followed for one year and has never recovered from her postoperative complete transverse myelitis.

NONGLIOMATOUS TUMORS

Melanoma.—Frazier and Allen (1918) listed 1 case of intramedullary melanoma, collected from the literature, in which the tumor was exposed at operation. No mention was made as to whether this was a primary or a metastatic lesion, and we have been unable to find the case

in our survey of the literature. Kernohan (1941), in a review of the cases of intramedullary tumors of the cord and filum terminale treated surgically at the Mayo Clinic, cited 3 cases of melanoepithelioma but made no mention of the origin of the tumor or of the outcome. Our case (case 31) was that of a woman aged 62 whose first symptom, one year before operation, was sudden, severe root pain radiating to the abdomen. Partial transverse myelitis developed at the level of the ninth dorsal segment. A dark, solid, infiltrating tumor was partially excised. The patient was given moderately intense roentgen ray therapy. After operation the transverse myelitis was complete, and the course was progressively downhill, the patient dying six months after operation. Careful clinical investigation revealed no source of metastasis. It can only be assumed, however, that this was a primary intramedullary melanoma since permission for autopsy was not obtained.

Dermoid Cyst.—Review of the literature reveals only 3 indisputable cases of dermoid of the spinal cord which were exclusively intramedullary. Sauerbruch and Hartmann (1935) reported a case of a girl of 13 in which a dermoid cyst located in the fifth dorsal segment was totally excised. The patient was reported as greatly improved three and a half years later. Love and Kernohan (1936) listed a case of dermoid cyst of the conus medullaris and filum terminale which was evacuated but not totally excised. In a personal communication to the authors, they stated that after operation the patient's condition remained unchanged and that his local physicians report that he is now worse (seven years after operation). Bradford (1938) reported a case of dermoid cyst of the conus in a boy of 3 years. Partial excision resulted in improvement, the period of observation being five years.

We wish to report 3 additional cases of intramedullary dermoid from this clinic. Two of these have previously been mentioned by List (1941) in connection with intraspinal epidermoid, dermoid and dermal sinus. In case 34 a girl aged 19 had pain in the lower part of the back three years prior to operation. Flaccid paresis gradually developed in both legs, without sensory or sphincter disturbances. She had tenderness of the upper lumbar portion of the spine and signs of meningeal irritation. Operation revealed an intramedullary dermoid cyst of the conus and epiconus, which extended caudad, displacing the caudal roots anteriorly. Intracapsular enucleation of the intramedullary portion was done, and the caudal portion was totally excised. Examination three years and two months after operation revealed that the child's condition was improved and she was walking almost normally (she had also undergone several orthopedic procedures). In case 33, a youth aged 19 had a history of intermittent weakness of the legs for thirteen years. Examination revealed spastic paraparesis of the legs, with diminution of all forms of sensation below the second dorsal dermatome. An intramedullary dermoid cyst, extending from the eighth cervical to the seventh dorsal segment, was partially excised. The patient gradually improved for five years, then began to have an increasingly severe return of his previous symptoms and incontinence of the bowel and bladder. A second operation was done six years and five months after the original one. Complete recurrence of an encapsulated dermoid cyst was observed, and the cyst was totally removed. The patient had a stormy postoperative course; the paraparesis and loss of sensation and of sphincter control were worse. A high, septic type of fever developed, the cause of which was not determined. He returned home, where his condition gradually grew worse, until he died, five months after operation. Autopsy revealed an old appendical rupture with abscess formation, which had not been diagnosed because of loss of sensation below the eighth cervical dermatome and the flaccid weakness of the abdominal musculature.

We wish to add a personal case, which we believe to be the first reported instance of multiple intramedullary dermoid cysts in which operation was performed.

CASE 35.—A woman aged 46 first noticed weakness of her left ankle twenty-six years prior to entrance to the hospital. "Claw foot" deformity developed on the left side. She then showed no progression of symptoms for about fifteen years, at which time she had a bout of pain in the lower part of the back, radiating down the distribution of the sciatic nerve bilaterally. After this she had similar attacks and began to have increasing weakness and atrophy of her left leg. Two years prior to entry "hammer toes" of the left foot developed, which were corrected by osteotomy. Shortly after this there was onset of urinary incontinence, which progressed to complete loss of bladder control. There was also gradual development of fecal incontinence. On her admission to the hospital neurologic examination revealed a conus syndrome with saddle anesthesia; spastic paraparesis, more pronounced on the left side; hyperactive patellar reflexes, and absence of the achilles jerk. There were no pathologic toe signs. Position and vibratory senses were normal. Roentgenograms of the spine revealed widening of the spinal canal at the dorsolumbar junction. Spinal puncture revealed no block; there was pronounced xanthochromia, and the protein of the spinal fluid was 222 mg. per hundred cubic centimeters. Myelographic examination was not made.

Laminectomy, by one of us (W. W. W.), was performed on May 10, 1941, at which time the dorsal spinous processes and laminas of the twelfth dorsal and the first, second and third lumbar vertebrae were re-

moved subperiosteally. Epidural fat was scanty, and the dural sac was enlarged. On incision of the dura, the cord was seen to be avascular, with a bulbous enlargement of the conus and epiconus. The contents of this area were aspirated, and thick, yellowish puslike material was obtained. The cord was incised longitudinally just to the right of the midline, and a large, well encapsulated cyst was encountered. This was evacuated of yellowish, cheeselike material, containing numerous hairs, and the capsule was entirely removed. At its upper margin was encountered a marble-sized, discrete cyst, which was also totally removed. There was no demonstrable connection between these two cysts. Several smaller yellowish, and also discrete, cysts were removed, the dorsal portion of the cord being laid wide open for a distance of 5 cm. In one area cartilaginous material, as well as an ill defined, loculated tissue, resembling skin, was encountered. This could not be removed entirely. Several cysts were matted together and their contents merely evacuated, their adherent walls being left intact. The dura was left open, with numerous cross cuts, so that adequate decompression was insured. Muscle and fascia were closed with steel wire sutures; the subcutaneous tissues, with surgical gut, and the skin, with black silk sutures. Convalescence was uneventful, the patient walking on the twentieth postoperative day.

The patient has been followed for two years and has shown excellent improvement. At the time of writing, she is free from her previous pain in the back and from her sciatic pain. Her bladder control is almost normal. She is slightly constipated and has fecal incontinence only with use of laxatives. The weakness of her legs has improved, and she is doing all her own housework. Neurologic examination reveals loss of superficial sensation below the fourth lumbar dermatome bilaterally. Vibration and position senses are absent over the right foot but normal over the left foot. The motor weakness and atrophy of the left leg seem slightly improved, but the gluteal muscles are definitely paretic on the left side. The patellar reflex is normal bilaterally. The achilles jerk is absent, and the plantar reflex is extensor on the left side and flexor on the right side.

Pathologic material removed at operation is characteristic of dermoid, the cyst walls containing skin, sebaceous glands, hair follicles, peripheral nerves and fibrocartilage.

The commonest location for intramedullary dermoid is the conus medullaris, although it may occur elsewhere. In all of our cases there was characteristic widening of the spinal canal at the site of the tumor. Clinically, aside from the long duration of symptoms, there is no characteristic or diagnostic sign to distinguish dermoid from other intramedullary lesions. The presence of a midline dimple or a frank dermal sinus, however, is certainly of diagnostic significance.

The ideal surgical treatment of dermoid cyst is total excision. This is frequently impossible, as demonstrated in the cases described. The degree of removal will depend on the judgment and skill of the surgeon. The rate of recurrence is slow, and a second operation is feasible. Roentgen ray therapy may be considered valueless.

We shall mention only in passing that cases of intramedullary epidermoid have been reported (for further particulars, see the articles by Boldrey and Elvidge and Falconer and Hooper).

Lipoma.—Our series contains no instance of a purely intramedullary lipoma as judged by the definition of Stookey, that is, without accompanying extramedullary tumor tissue or the deformities of spina bifida. For a comprehensive review of the literature we recommend the article by Bucy and Gustafson.

Tuberculoma.—We found in the literature 7 cases of tuberculoma exposed surgically. We have no such case in our series. The first report was by Krauss and McGuire, in 1909. The literature has been reviewed by Fiamberti (1929), and additional cases have been reported by von Eiselsberg (1932) and de Quervain (1926). The incidence of intramedullary tuberculoma appears to be increasingly rare.

Intramedullary Blood Clot.—Although this lesion cannot be considered a true tumor, we have included our case here because we were unable to discover a similar instance reported in the past. Elsberg and Beer, in 1911, mentioned a case of intramedullary hemorrhage which they concluded had probably taken place into a cystic glioma or a cavity of syringomyelia. This clot was treated by the method of extrusion, with considerable improvement. In our case, however, the hemorrhage was not into a previously existing cavity but, rather, into the substance of the cord from a defective intramedullary vessel.

Case 32.—A man aged 22 entered the hospital complaining of a "stroke." For the past two years he had had brief periods of aching pain and slight weakness of the upper portion of the right arm. One year prior to entry he had sudden onset of aching pain in the region of the right triceps muscle during intercourse. Within a few seconds his entire right arm and hand became numb and weak; then progressive numbness and weakness spread down his body and involved both lower extremities. His left arm alone remained normal. His right hand and arm were weakest, but the left side of the trunk and the left leg were most numb. He had retention of urine. The following day his symptoms began gradually to recede and cleared entirely within a week except for residual burning and slight weakness of the right hand. One month prior to entry he had another attack, unrelated to effort. He had onset of pain in the right arm and then became completely paralyzed and anesthetic below the neck. His condition began to improve the following day, but he still had, on entrance to the hospital, definite weakness of the right side of the body, more pronounced in the arm than in the leg, and numbness of the left arm and leg, particularly the leg. He had urinary frequency and occasional incontinence.

Neurologic examination revealed no abnormality of the cranial nerves. There was a partial Brown-Séquard syndrome, with flaccid weakness and minimal atrophy of

the right forearm and hand and spastic weakness of the right leg. Motor power on the left side was normal. There were diminution of pain and temperature sense, but not of touch sense or of perception of skin writing, below the first dorsal dermatome on the left side. Vibration and position senses were intact throughout. No sensory changes were noted on the right side. The deep reflexes of the upper part of the right arm were diminished and were normal in the left arm. Abdominal reflexes were absent on the right side and were diminished on the left. The deep reflexes were increased, with ankle clonus and extensor plantar response in the right leg; those of the left leg were normal.

A clinical diagnosis of hematomyelia was made. Routine roentgenograms of the spine disclosed no abnormality. Spinal puncture revealed no block. The fluid was clear and colorless; the protein content was 53 mg. per hundred cubic centimeters. A myelogram revealed nothing significant.

At operation (Jan. 23, 1943), performed by one of us (W. W. W.), the cervicodorsal junction was exposed by laminectomy of the sixth and seventh cervical and first dorsal vertebrae. There was a normal amount of epidural fat; the dura also appeared normal. The cord, however, revealed slight pallor and bulging at approximately the level of the seventh and eighth cervical and first dorsal segments. There was little pulsation of the cord at this level. Aspiration of the cord revealed nothing. A vertical myelotomy, with the incision slightly to the right of the midline, revealed the cord to be slightly more yellow than normal at the depth of about 2 mm., and immediately below this appeared a discrete, apparently encapsulated, dark red tumor. By gentle retraction of the incised cord the entire mass was exposed over its length, of 2 cm. At the upper pole was a large, tortuous, pulsating vessel, which was clipped, coagulated and divided. The tumor was then easily lifted out of its intramedullary bed and removed entirely. The surrounding cord tissue was yellow and slightly softened. The operative diagnosis was ependymoma. The dura was closed with a running black silk stitch; the muscles and fascia, with steel wire; the subcutaneous tissues, with surgical gut, and the skin, with black silk. Postoperative convalescence was uneventful. The patient was out of bed and walking on the fourteenth postoperative day. He was discharged after two weeks of active and passive physical therapy. Examination at the time of discharge revealed increased weakness in the grip of the right hand and mild spasticity and weakness of both legs, more pronounced on the right side. Vibration and position sense were preserved on the left and were absent on the right. Bladder control also was similar to that before operation. The patient has not been seen since but writes that sensory loss on the left side is the same as before operation. He is driving his car and doing light work about the house. Since his bladder control is better and he is free from pain, he feels that his condition is improved over that before operation, five months ago.

Pathologic examination of the mass revealed it to be a discrete blood clot, measuring approximately 2 by 0.75 cm. The accompanying artery showed thickening and sclerosis of the walls. The smaller arteries also showed changes.

We believe this to be the first reported case of total and successful removal of an intramedullary blood clot arising from rupture of an abnormal vessel, unassociated with cavitation of the cord.

Syringomyelia. — Elsberg (1912) is credited with the first description of a case of syringomyelia treated surgically, having mentioned the possibility in 1911. In the literature, however, the method of incision of the syringomyelic cavity is known as the Puusepp method (1926). Outstanding reviews were published by Juźelevskij, in 1930 (6 personal cases); Peiper, in 1931 (4 cases); Frazier and Rowe, in 1936 (5 cases), and Adelstein, in 1938 (3 cases). The last author collected from the literature 120 cases of syringomyelia treated surgically. The general opinion expressed by most authors is that surgical intervention is indicated, not with hope of cure but for the purpose of arresting, for a time at least, the progress of the disease. Frazier and Rowe, and Adelstein concluded that about 50 per cent of the patients undergoing operation are sufficiently improved to return to work. We present the results in 20 cases in which operation was performed in this clinic. These results offer less opportunity for optimism regarding the surgical treatment of syringomyelia.

Of the 20 patients, 11 were males and 9 females. The youngest patient was 16 and the oldest 55 years of age; the average age was 35 years and 4 months. In most cases (12, or 60 per cent) the cavity was in the cervicodorsal region. In 5 cases it was in the cervical region; in 1, in the dorsal region, and in 2, in the conus medullaris. The shortest duration of symptoms, with rapidly developing flaccid paraparesis, was ten days. The longest duration of symptoms was twenty-one years, the disturbance starting with numbness and pain in the left arm and left leg and progressing to mild tetraparesis. The average duration of symptoms was five and a half years. Clinical examination revealed no significant variations in the classic description of the disease. In 5 cases there was concomitant scoliosis; in 4 cases out of 13 partial or complete manometric block was apparent on lumbar puncture.

Only one type of operation was performed, that is, as extensive as possible a vertical incision of the syringomyelic cavity. No attempt was made to insert foreign material in order to prevent subsequent closure. In 2 cases a second operation was done, one year and three years respectively after the first operation. In both cases the cavity had been reenclosed.

In 1 case death occurred five days after operation, of septicemia arising from a previously infected hand. In 8 other cases the patients subsequently died. The shortest period of survival was one month and the longest nine years.

The average length of follow-up observation was three years and six months. In 3 cases the patient died in less than one year, and in 1 case the patient has not been heard from since discharge from the hospital. In the remaining 16 cases the patients have been followed for one year or more, the longest period being thirteen years. Of the 16 cases in which the follow-up period was one year or longer, improvement occurred in 2. In 1 case, a woman aged 48 was followed for only one year. Sensation, motor power and sphincter control were improved. The lesion, however, was of the conus, and doubt exists as to whether it was true syringomyelia or cystic tumor. The specimens of cyst wall examined histologically, however, revealed no evidence of tumor. In the other case a man aged 40 sustained a compression fracture of the first lumbar vertebra in a fall. He had immediate flaccid weakness of his legs and incontinence of urine and feces. The weakness of his legs improved slightly, but his incontinence continued. On admission to the hospital neurologic examination revealed a typical conus syndrome. Operation dislosed a swelling of the conus, which was incised, with exposure of a smooth-walled cyst containing yellowish fluid. There was no evidence of tumor. The patient has now survived thirteen years after operation. His legs are stronger, and he can walk about with a cane. He is no longer completely incapacitated and is able to work at a clerical job and help about the farm. Bladder and bowel control are slightly improved. In the remaining 14 cases there were typical syringomyelic cavities. In 2 cases the condition was unchanged, and in 12 it became worse. In 5 of these 12 cases the patients died. It should be added that in 6 of the 20 cases in this series roentgen ray therapy was given. Of these, the condition in 1 is unchanged and that in 5 worse (3 patients have died).

From our material only one conclusion can be reached, namely, that simple incision of a syringomyelic cavity offers little lasting benefit. It is possible that prevention of reclosure of the cavity might alter this poor prognosis.

GENERAL CONCLUSIONS

In a clinicopathologic presentation of 68 consecutive cases of intramedullary lesions treated surgically, emphasis is placed on the clinical results following operation. No definite conclusions can be reached from such a limited number of cases, but certain tentative statements may be made:

1. The surgical treatment of intramedullary tumors of the cord produces unexpectedly good results when the location and size, and often the infiltrative character, of these neoplasms are taken into consideration. The very nature of the spinal cord, a vulnerable structure enclosed in an unyielding bony canal, makes these results even more surprising.

2. The comparatively slow growth of these tumors offers promise of better results in the future, with the attainment of early diagnosis and with surgical intervention before irreversible damage to the spinal cord has occurred.

3. The best surgical results are obtained when the tumor can be totally excised, without damage to the cord. If this is not possible because of lack of demarcation, decompressive vertical incision of the posterior surface of the cord over the entire extent of the tumor should be attempted. The concept that an intramedullary tumor encountered at operation is inoperable and is best left alone, or only a specimen taken for biopsy, is unjustifiable.

4. Intensive roentgen ray therapy following operation appears to be of benefit with certain gliomas of the spinal cord.

5. Ependymoma, because of its frequent demarcation and the apparent limitation of its growth, offers a better prognosis than the more invasive gliomas.

6. Intramedullary dermoid, because of its slow growth and encapsulation, lends itself either to total or to partial excision.

7. The surgical treatment of syringomyelia, with or without roentgen ray therapy, has not produced the hopeful results previously reported in the literature.

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BIBLIOGRAPHY

Abrahamson, I., and Climenko, H.: Symptomatology of Spinal Cord Tumors, J. A. M. A. **75**:1126-1132 (Oct. 23) 1920.

Adams, D. S.: Glioma of Lumbar Cord: Case Report, Boston M. & S. J. **186**:738-740 (June 1) 1922.

*Adelstein, L. J.: Surgical Treatment of Syringomyelia, Am. J. Surg. **40**:384-395 (May) 1938.

——and Paterson, G. H.: Surgical Treatment of Ependymal Glioma of the Spinal Cord, Arch. Surg. **30**:997-1014 (June) 1935.

Allen, I.: Glioma of the Cervical Cord, Canad. M. A. J. **28**:417-419 (April) 1933.

Allen, S. S., and Kahn, E. A.: Case of Scoliosis Produced by Spinal Cord Tumor, J. Nerv. & Ment. Dis. **77**:53-55 (Jan.) 1933.

Alurralde, M., and Sepich, M. J.: Tumor inframedular del cono y epicono, Prensa méd. argent. **22**:1-6 (Jan. 2) 1935.

Arcé, J., and Balado, M.: Diagnostic et traitement des tumeurs intra-rachidiennes, Cong. Soc. internat. de chir., Rap. **2**:825-839, 1932.

Auerbach, S.: Ueber einen bemerkenswerten fall von intramedullärem Rückensmarkstumor, J. f. Psychol. u. Neurol. **17**:159-168, 1910.

Bailey, Pearce: Surgical Diagnosis and Treatment of Tumors in and About the Spinal Cord, J. A. M. A. **54**:849-853 (March 12) 1910.

Spinal Cord Tumor and Trauma: A Report of Two Cases, J. Nerv. & Ment. Dis. **35**:315-319, 1908.

Bailey, P., and Bucy, P. C.: Tumors of the Spinal Canal, S. Clin. North America **10**:233-257 (April) 1930.

Baker, G. S., and Statford, D. E.: Complete Removal of a Large Intramedullary Tumor (Ependymoma) from the Cervical Portion of the Spinal Cord: Report of Case, Proc. Staff Meet., Mayo Clin. **16**: 421-423 (July 2) 1941.

Beckman, E. H.: Tumors of the Spinal Cord, with a Report of Eighteen Cases, Journal-Lancet **37**:35-42 (Jan. 15) 1917.

Bériel, L.: Tumeurs intrarachidiennes sur l'opportunité des interventions, Lyon méd. **132**:66-67 (Jan. 10) 1923.

——and Mestrallet, A.: Tumeurs intrarachidiennes: Un cas de gliomatose intra et extra-medullaires (projections), ibid. **142**:713-716 (Dec. 16) 1928.

Boldrey, E. B., and Elvidge, A. R,: Dermoid Cysts of the Vertebral Canal, Am. J. Surg. **110**:273-284 (Aug.) 1939.

*Bradford, F. K.: Intramedullary Dermoid Cyst, Ann. Surg. **107**:107-115 (Jan.) 1938.

*Bregman, L.: Ein Beitrag zur Klinik und zur operativen Behandlung der Rückenmarks Geschwülste, Deutsche Ztschr. f. Nervenh. **31**:68-85 (Sept.) 1906.

*Browder, J.: Tumors of the Spinal Cord, Am. J. Surg. **24**:1-10 (April) 1934.

Brun, H.: Ueber einen zweiten Fall von operativer Entfernung eines subpial gelegenen Rückenmarkstumors, Deutsche Ztschr. f. Chir. **110**:487-506 (July) 1911.

Bucy, P. C., and Gustafson, W. A,: Intradural Lipoma of the Spinal Cord, Zentralbl. f. Neurochir. **3**:341-349 (Jan.) 1939.

*Cairns, H., and Riddoch, G.: Observations on the Treatment of Ependymal Gliomas of the Spinal Cord, Brain **54**:117-146 (June) 1931.

Cambier, P., and Ley, R. A.: Tumeur médullaire, J. belge de neurol. et de psychiat. **34**:321-325 (May) 1934.

Christophe, L.: Tumeur intramédullaire chez une enfant de moins de trois ans; extirpation; guérison; classification histologique difficile, Rev. neurol. **2**:517-522 (Nov.) 1932.

*Critchley, M., and Ferguson, F. R.: Cerebrospinal Epidermoids (Cholesteatomata), Brain **51**:334-384 (Oct.) 1928.

Cushing, H.: The Intracranial Tumors of Preadolescence, Am. J. Dis. Child. **33**:551-584 (April) 1927.

Delagénière, Y.: Considérations sur le pronostic des tumeurs intra-rachidiennes opérées, Cong. Soc. internat. de chir., Rap. **2**:868-878, 1932.

Dercum, F. X., and Da Costa, J. C.: Intramedullary Tumor of Cervical Cord: Diagnosis; Removal in Two-Stage Operation; Gradual Improvement, J. Nerv. & Ment. Dis. **44**:97-103 (Aug.) 1916.

*Dyke, C. G., and Davidoff, L. M.: Roentgen Treatment of Diseases of the Nervous System, Philadelphia, Lea & Febiger, 1942.

von Eiselsberg, A.: Intramedulläre Rückenmarkstumoren, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **42**:613-622, 1932.

——and Marburg, O.: Zur Frage der Operabilität intramedullärer Rückenmarkstumoren, Arch. f. Psychiat. **59**:453-461 (Sept.) 1918.

——and Ranzi, E.: Ueber die chirurgische Behandlung der Hirn- und Rückenmarkstumoren, Arch. f. klin. Chir. **102**:309-468 (Sept.) 1913.

Elsberg, C. A.: Surgery of Intramedullary Affections of the Spinal Cord: Anatomic Basis and Technic, J. A. M. A. **59**:1532-1536 (Oct. 26) 1912.

Tumors of the Spinal Cord, New York, Paul B. Hoeber, 1925.

The Diagnosis and Surgical Treatment of Tumors of the Spinal Cord, Cong. Soc. internat. de chir., Rap. **2**:385-438, 1932.

Surgical Diseases of the Spinal Cord, Membranes and Nerve Roots: Symptoms, Diagnosis and Treatment, New York, Paul B. Hoeber, Inc., 1941.

——and Beer, E.: The Operability of Intramedullary Tumors of the Spinal Cord, Am. J. M. Sc. **142**:636-647 (Nov.) 1911.

——and Dyke, C. G.: The Diagnosis and Localization of Tumors of the Spinal Cord by Means of Measurements Made on the X-Ray Films of the Vertebrae, and Correlation of Clinical and X-Ray Findings, Bull. Neurol. Inst. New York **3**:359-394 (March) 1934.

Falconer, M. A., and Hooper, R. S.: Intramedullary Epidermoid Cyst of the Spinal Cord: Report of a Case with Remarks on Early Diagnosis, Brit. J. Surg. **28**:538-543 (April) 1941.

Feiling, A.: Two Cases of Intramedullary Tumor of the Spinal Cord, with Operation, Lancet **1**:957-960 (May 1) 1920.

*Fiamberti, A. M.: Tuberculoma solitario del midollo spinale, Riv. di pat. nerv. **34**:617-671 (Nov.) 1929.

Foerster, O.: Report of Cases of Spinal Cord Tumors at the Breslau Surgical Society, Zentralbl. f. Chir. **56**:891-892 (April 6) 1929.

——and Bailey, P.: Gliomas of the Spinal Cord, in Jubilee Volume for Davidenkow, Leningrad, State Institute for the Publication of Biologic and Medical Literature, 1936 (not available).

Fraser, J.: A Cystic Dermoid Tumor of the Spinal Cord, Surg., Gynec. & Obst. **51**:162-168 (Aug.) 1930.

Frazier, C. H., and Allen, A. R.: Surgery of the Spine and Spinal Cord, New York. D. Appleton and Company, 1918.

——and Rowe, S. N.: The Surgical Treatment of Syringomyelia, Ann. Surg. **103**:481-497 (April) 1936.

——and Spiller, W. G.: Analysis of Fourteen Consecutive Cases of Spinal Cord Tumors, Arch. Neurol. & Psychiat. **8**:455-501 (Nov.) 1922.

*Globus, J. H., and Doshay, L. J.: Venous Dilatations and Other Intraspinal Vessel Alterations, Including True Angiomata with Signs and Symptoms of Cord Compression, Surg., Gynec. & Obst. **48**:345-366 (March) 1929.

Gross, S. W.: Concerning Intraspinal Dermoids and Epidermoids, with Report of a Case, J. Nerv. & Ment. Dis. **80**:274-284 (Sept.) 1934.

*Hamby, W. B.: Tumors in the Spinal Canal in Childhood: Analysis of Literature with Report of Case, J. Nerv. & Ment. Dis. **81**:24-42 (Jan.) 1935.

Heymann: Chordoma praesacrale, Klin. Wchnschr. **4**:899 (April 30) 1925.

*Horrax, G., and Henderson, D. G.: Encapsulated Intramedullary Tumor Involving the Whole Spinal Cord from Medulla to Conus: Complete Enucleation with Recovery, Surg., Gynec. & Obst. **68**: 814-819 (April) 1939.

Ingebrigtsen, R., and Leegaard, T.: Final Results in a Series of Operated Intraspinal Tumors, Acta chir. Scandinav. **82**:271-281, 1939.

*Ingraham, F.: Intraspinal Tumors in Infancy and Childhood, Am. J. Surg. **39**:342-376 (Feb.) 1938.

*Jabotinski, J.: Fibrolipome intradural de la moelle, Rev. neurol. **72**:15-31 (July) 1939.

*Juzelevskij, A.: Die chirurgische Behandlung der Syringomyelie nach Pussep, Beitr. z. klin. Chir. **148**:389-417, 1930.

Kernohan, J. W.: Primary Tumors of the Spinal Cord and Intradural Filum Terminale, in Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, pp. 991-1025.

Tumors of the Spinal Cord, Arch. Path. **32**:843-883 (Nov.) 1941.

——Woltman, H. W., and Adson, A. W.: Intramedullary Tumors of the Spinal Cord: Review of Fifty-One Cases, with an Attempt at Histologic Classification, Arch. Neurol. & Psychiat. **25**:679-701 (April) 1931.

Gliomas Arising from the Region of the Cauda Equina, ibid. **29**:287-307 (Feb.) 1933.

Krause, F.: Erfahrungen bei 26 operativer Fälle Rückenmarkstumoren. Rückenmarksoperationen mit Projectionen, Deutsche Ztschr. f. Nervenh. **36**: 106-113, 1908.

Surgery of Brain and Spinal Cord, translated by H. A. Haubold, New York, Rebman Co., 1912, vol. 3, pp. 1127-1132.

Krauss, W. C.: Three Cases of Spinal Cord Tumor Observed Within a Period of Ten Days: Observation on the Behavior of the Cerebrospinal Fluid, J. Nerv. & Ment. Dis. **37**:222-236 (April) 1910.

——and McGuire, E. R.: Intramedullary Tuberculoma Removed at the Level of the Fifth Thoracic Segment of the Spinal Cord, J. A. M. A. **53**:1911-1912 (Dec. 4) 1909.

*von Lennep, H.: Ueber Rückenmarkstumoren, Deutsche Ztschr. f. Chir. **160**:137-140 (Nov.) 1920.

Liedberg, N.: Zur Frage der Klinik und Therapie der Rückenmarkstumoren. Durch eine Reihe operierter Fälle beleuchtet, Acta chir. Scandinav. **77**:452-504, 1936.

*List, C. F.: Intraspinal Epidermoids, Dermoids and Dermal Sinuses, Surg., Gynec. & Obst. **73**:525-538 (Oct.) 1941.

*Love, J. G., and Kernohan, J. W.: Dermoid and Epidermoid Tumors (Cholesteatomas) of Central Nervous System, J. A. M. A. **107**:1876-1883 (Dec. 5) 1936.

McLean, A. J.: Spinal Tumors, with Report of Six Cases, West. J. Surg. **43**:1-15 (Jan.) 1935.

*Mahoney, W.: Die Epidermoide des Zentralnervensystems, Ztschr. f. d. ges. Neurol. u. Psychiat. **155**:416-471, 1936.

de Martel, T.: Chirurgie de la moelle et des racines rachidiennes, Bull. et mém. Soc. nat. de chir. **50**: 532-537 (April) 1924.

*Melinikoff-Razvedenkoff, N. F.: Ueber epidermoide und dermoide Cholesteatoma des Grosshirns und Rückenmarks mit besonderer Berücksichtigung der in der Ukraine beobachteten Fälle, Virchows Arch. f. path. Anat. **279**:702-723, 1931.

Michelsen, J.: Cholesteatom des Rückenmarks, Deutsche Ztschr. f. Nervenh. **127**:123-130 (July 9) 1932.

*Moersch, F. P.; Love, J. G., and Kernohan, J. W.: Melanoma of the Central Nervous System, J. A. M. A. **115**:2148-2155 (Dec. 21) 1940.

Naffziger, H. C., and Jones, O. W.: Dermoid Tumors of the Spinal Cord: Report of Four Cases, with Observations on a Clinical Test for Differentiation of the Source of Radicular Pains, Arch. Neurol. & Psychiat. **33**:941-958 (May) 1935.

Nonne: Weitere Erfahrungen an operiertem Fällen von Rückenmarkstumoren, Berl. klin. Wchnschr. **50**:941 (May 19) 1913.

Peiper, H.: Operation oder versuchsweise Bestrahlung der Rückenmarkstumoren? Deutsche med. Wchnschr. **53**:1627-1630 (Sept. 23) 1927.

Die operative Behandlung der Syringomyelie, Nervenarzt **4**:436-453 (Aug. 15) 1931.

*Potel, G., and Veaudeau: La chirurgie des tumeurs du rachis et de la moelle, Rev. de chir. **48**:477-520, 1913.

Puusepp, L.: Traitement opératoire dans deux cas de syringomyelie; amélioration notable, Rev. neurol. **1**:1171-1179 (June) 1926.

Diagnostic et traitement chirurgical des tumeurs de la moelle, Arch. franco-belges de chir. **28**:89-124 (Feb.) 1925.

de Quervain, F.: Zur Klinik und Operation der intramedullären Rückenmarkstumoren, Schweiz. med. Wchnschr. **56**:585-588 (June 19) 1926.

Randi, E.: Operationen wegen Rückenmarkstumor und ihre Resultate, Arch. f. klin. Chir. **120**:489-517 (Sept. 5) 1922.

——and Sgalitzer, G., Jr.: Ueber die Ergebnisse unserer Operationen wegen Rückenmarkstumor, Wien. klin. Wchnschr. **50**:777-780 (May 22) 1937.

Rasmussen, T. B.; Kernohan, J. W., and Adson, A. W.: Pathologic Classification, with Surgical Consideration, of Intraspinal Tumors, Ann. Surg. **111**:513-530 (April) 1940.

*Robineau: Diagnostic et traitement des tumeurs de la moelle, Cong. Soc. internat. de chir., Rap. **2**:575-655, 1932.

Röpke, W.: Ueber die operative Entfernung intramedullärer Rückenmarkstumoren, Arch. f. klin. Chir. **96**:963-980 (Dec. 21) 1911.

Rothmann, M.: Gegenwart und Zukunft des Rückenmarkschirurgie, Berl. klin. Wchnschr. **50**:598-603 (March 31) 1913.

Sachs, E., and Fincher, E. F., Jr.: Intramedullary Lipoma of the Spinal Cord: Complete Operative Removal, Arch. Surg. **17**:829-833 (Nov.) 1928.

Saucier, J., and Trepagnier, F. D.: Syndrome de Brown-Séquard: Blocage sous-arachnoïdien. Manifestations imprévues dues au lipiodol. Laminectomie bilatérale de CI à LI. Gliome medullaire. Radiotherapie intensive. Amélioration, Union méd. du Canada **64**:800-807 (July) 1935.

Sauerbruch, F., and Hartmann, F.: Beitrag zur Chirurgie intramedullärer Neubildungen, Schweiz. med. Wchnschr. **65**:26-28 (Jan. 12) 1935.

——and Thiele, R.: Zwei erfolgreich operierte intramedulläre Rückenmarkstumoren, Acta chir. Scandinav. **72**:431-440, 1932.

Schaeffer, H.: Radiotherapie des tumeurs medullaires, Presse méd. **33**:1620-1622 (Dec. 9) 1925.

Schlesinger, H.: Beiträge zur Klinik der Rückenmarks und Wirbeltumoren, Jena, G. Fischer, 1898 (not available).

Schmieden, V., and Peiper, H.: Ueber ein erfolgreich operiertes endomedullares Lipom des Halsmarks, nebst einigen Bemerkungen zur Chirurgie der intramedullaeren Tumoren, Deutsche med. Wchnschr. **55**:513-516 (March 29) 1929.

Schönbauer, L.: Die Ergebnisse der operativen Behandlung der Rückenmarkstumoren, Arch. f. klin. Chir. **154**:645-692, 1929.

Shallow, T. A.: Four Cases Illustrating Symptoms, Diagnosis and Treatment of Spinal Cord Compression, S. Clin. North America **8**:885-899 (Aug.) 1928.

Sicard, J. A., and Haguenau, J.: L'image lipiodolée sous-arachnoidienne, en ligne fastonée longitudinale, des tumeurs intra-medullaires, Rev. neurol. **1**:676-678 (May) 1925.

*Spurling, R. G., and Mayfield, F. H.: Neoplasms of the Spinal Cord: A Review of Forty-Two Surgical Cases, J. A. M. A. **107**:924-929 (Sept. 19) 1936.

Steinke, C. R.: Spinal Tumors: Statistics on a Series of Three Hundred and Thirty Collected Cases, J. Nerv. & Ment. Dis. **47**:418-426 (June) 1918.

*Stookey, B.: Tumors of the Spinal Cord in Childhood, Am. J. Dis. Child. **36**:1184-1203 (Dec.) 1928.

Intradural Spinal Lipoma: Report of a Case and Symptoms for Ten Years in a Child Aged Eleven, Arch. Neurol. & Psychiat. **18**:16-43 (July) 1927.

Tarlov, I. M.: Ependymoma of the Filum Terminale, Arch. Neurol. & Psychiat. **32**:1045-1054 (Nov.) 1934.

*Trachtenberg, M. A.: Ein Beitrag zur Lehre von dem arachnoidealen Epidermoiden und Dermoiden des Hirns und Rückenmarks, Virchows Arch. f. path. Anat. **154**:274-291, 1898.

Veraguth, O., and Brun, H.: Subpialer, makroskopisch intramedullärer Solitärtuberkel in der Höhe des vierten und fünften Cervicalsegmentes; Operationen; Genesung, Cor.-Bl. f. schweiz. Aerzte **40**: 1097-1109 (Nov. 20) 1910.

Vincent, C.: La chirurgie des tumeurs intramédullaires en France en 1913. Sur un cas rapporte par Gendron, opéré avec succès par Th. de Martel, Rev. neurol. **1**:491-502 (April) 1927.

Walker, A. E.; Jessico, C. M., and Marcovich, A. W.: The Myelographic Diagnosis of Intramedullary Spinal Cord Tumors, Am. J. Roentgenol. **45**:321-331 (March) 1941.

Weaver, J. C.: Intramedullary Tumors, J. M. A. Georgia **25**:61-63 (Feb.) 1936.

Woods, A. H.: Removal of a Tumor from the Spinal Cord in Syringomyelia, Arch. Neurol. & Psychiat. **20**:1258-1268 (Dec.) 1928.

*Additional bibliographic references are included.

CEREBROSPINAL FLUID PRESSURE UNDER CONDITIONS EXISTING AT HIGH ALTITUDES

A CRITICAL REVIEW

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The literature contains conflicting data on the effect of altitude on intracranial pressure. If during the air transportation of men with head injuries the intracranial pressure should be increased, the results might be deleterious to the condition of the patients. Therefore we propose in this paper to examine the available reports which are relevant to this subject, with a view to drawing conclusions as to what changes, if any, may be expected and under what circumstances they may occur.

High altitude results in the following changes in the physical environment of the body: first, reduced barometric pressure; second, reduced tension of oxygen, and third, reduced temperature. There is no change in the percentage composition of the atmospheric gases.

In order to examine the effect of altitude on cerebrospinal fluid pressure, one must consider how the aforementioned changes, produced by high altitudes, would affect the well recognized mechanisms by which cerebrospinal fluid pressure may be altered. These mechanisms have been summarized by Ayer [1] as follows: 1, hydrostatic pressure, when the subject is in any position except the horizontal; 2, brain bulk, or the amount of solid substance in the cranium; 3, secretion pressure of the cerebrospinal fluid; 4, rate of absorption of the cerebrospinal fluid; 5, elasticity of the dura; 6, intracranial arterial pressure, and 7. intracranial venous pressure.

For obvious reasons, the first factor listed need not be discussed further in the present study. It is proposed to consider the work of other investigators on the remaining factors so far as their results are related to the present problem.

Since the reduced tension of oxygen at high altitudes is known to have a striking effect on the organism, one may feel justified in examining carefully the effects of lack of oxygen as such on some of the mechanisms which alter cerebrospinal fluid pressure with a view to coordinating such information with investigations carried out on the cerebrospinal fluid at high altitudes. This is further made necessary because in some of the investigations on cerebrospinal fluid at high altitudes no attempt has been made to examine the factor of anoxia quantitatively. Hence, in the discussions which follow, the various mechanisms which are known to alter cerebrospinal fluid pressure will be considered from the point of view of anoxia itself and anoxia due to reduced atmospheric pressure, as well as some of the secondary effects of reduced barometric pressure. Finally, it is proposed to consider the available reports in which cerebrospinal fluid pressure was determined at conditions of high altitude, with a view to assessing the significance of the results and conclusions as they have been obtained by the various authors.

The effect of temperature will not be considered because significant changes in temperature can be adequately prevented by special clothing and aircraft cabin construction. Further, any effect that reduced temperature might have on cerebrospinal fluid pressure would lack significance by comparison with the effect it would have on the body as a whole if the reduced temperature were allowed to act without interference.

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1. Ayer, J. B.: Cerebrospinal Fluid Pressures from the Clinical Point of View, A. Research Nerv. & Ment. Dis., Proc. **4**:159, 1926.

This report is limited to an examination of intracranial pressure on acute exposures to high altitude. It does not take into consideration the behavior of intracranial pressure in persons accustomed to living for long periods at high altitudes. Presumably, the factors of adjustment in such persons would obviate the maintenance of any state of chronically abnormal intracranial pressure.

FACTORS INFLUENCING CEREBROSPINAL FLUID PRESSURE DURING ANOXIA

Brain Bulk, or Amount of Solid Substance in the Cranium.—Several investigators have independently shown that there is an increase in the bulk of the cerebral tissues under conditions of anoxia. White, Verlot, Selverstone and Beecher [2] observed that cats under anesthesia induced with pentobarbital sodium, and also cats under ether anesthesia, when they were breathing low oxygen mixtures, showed an increase in the intercellular or the intracellular fluid of the brain, leading to an increase in the size of the organ. However, there was no correlation between the cerebrospinal fluid pressure as recorded in the cisterna magna and the degree of swelling of the cerebral tissues during uncomplicated anoxia. In certain of the experiments there was a rapid rise in the cerebrospinal fluid pressure with the onset of anoxia, but often the pressure in the final reading had fallen to a normal, or nearly normal, level. In the majority of cases the final pressure was low. In other cases the initial increases failed to occur. The extent of change in pressure was in no case remarkable. The maximum change in pressure recorded when the animal was under pentobarbital anesthesia with anoxia was 112 mm. of water, and this value was obtained in the case of only 1 cat. The authors postulated mechanisms for adjustment of pressure to account for their observations and the compatibility of their results with the Monro-Kellie hypothesis. This subject will be referred to again.

Wolff and Lennox,[3] employing cats which were under barbiturate anesthesia and had been prepared by means of Forbes's window in the skull and cisternal puncture, investigated the effect of anoxemia. They produced progressive decreases in the oxygen content of the inspired air by gradually increasing the proportion of nitrogen. In an experiment which was recorded in detail, the oxygen saturation of the blood fell from 89 per cent in the control period, to successive concentrations of 11, 54, 76 and 93 per cent of the initial value. There was also a slight decrease in the percentage of carbon dioxide in the blood, the decrease reaching a maximum of 15 per cent by the end of the experiment. The authors observed a progressive increase in the cerebrospinal fluid pressure, the pressure rising from 75 to 100 mm. of isotonic solution of three chlorides U. S. P., independent of any notable change in the arterial blood pressure. Toward the end of the experiment they observed an increase of 7 per cent in the size of the pial arteries. Terminally, there was a sharp drop in both the cerebrospinal fluid and the arterial blood pressure coincident with deterioration of the animal. Such an increase in the volume of the intracranial arterial tree cannot alone be considered responsible for such an increase in intracranial pressure. Some other factor, such as increase in the bulk of the brain or changes in the production of cerebrospinal fluid, must be postulated. The authors did not attempt to investigate further the cause of this increase in pressure.

Michelson and Thompson [4] studied human subjects in a chamber in which a simulated altitude of 15,500 to 17,000 feet (9,400 to 10,000 meters) was obtained by addition of nitrogen to the atmospheric air; i.e., the partial pressure of oxygen was reduced, and the percentage composition of the air was altered. The subjects remained under these conditions for from four to seven hours. The oxygen saturation of the blood varied from 50 to 70 per cent, the amount depending on the individual studies, although the atmospheric conditions were the same. Under these conditions clinical pictures were produced consisting of headache, projectile vomiting, bradycardia, semiconsciousness, disturbances of respiration and mental impairment. The authors concluded that these symptoms justified a diagnosis of increased intracranial pressure, although no actual determinations of pressure were made. If such a condition was present an increase in the bulk of the brain must have contributed in large measure, and it is significant that these symptoms were initially aggravated by the giving of atmospheric air with sufficient oxygen to raise the oxygen in the blood to the normal level, an effect indicating that a not easily reversible change had occurred in the intracranial contents which prolonged the

2. White, J. C.; Verlot, M.; Selverstone, B., and Beecher, H. K.: Changes in Brain Volume During Anesthesia: The Effects of Anoxia and Hypercapnia, Arch. Surg. **44**:1-21 (Jan.) 1942.

3. Wolff, H. G., and Lennox, W. G.: Cerebral Circulation: The Effect on Pial Vessels of Variations in Oxygen and Carbon Dioxide Content of the Blood, Arch. Neurol. & Psychiat. **23**:1097 (June) 1930.

4. Michelson, J., and Thompson, J. W.: Oxygen Want and Intracranial Pressure, Am. J. M. Sc. **195**: 673, 1938.

existence of this syndrome. In the light of other investigations, this change was most probably some degree of cerebral edema.

One might mention, in considering this experiment, that Haldane and Priestley [5] stated that anoxemia is more effectively produced by reducing the percentage of oxygen in the air breathed at normal atmospheric pressure than by lowering the partial pressure of oxygen by means of reduction of the total barometric pressure, the percentage composition of the air remaining constant. This is attributed to the fact that the rate of diffusion of a gas increases with a fall of barometric pressure because the mean free path of each molecule before it strikes another is increased. This facilitates the progress of oxygen molecules toward the alveolar epithelium and presumably compensates to some extent for the fall in alveolar oxygen pressure which occurs at low barometric pressures. The true significance of this point would probably not be of great moment.

Maurer,[6] experimenting with cats under anesthesia induced with pentobarbital sodium supplemented with curare, determined the cerebrospinal fluid pressure by cisternal puncture while the animals breathed atmospheres containing 6 and 8 per cent oxygen. In an experiment in which the cat was exposed to 8 per cent oxygen for fifty-two minutes, there was an immediate rise in cerebrospinal fluid pressure with a concomitant increase of arterial blood pressure of short duration. The blood pressure then rapidly diminished. However, the cerebrospinal fluid pressure continued to increase until fifteen minutes before death, when there was a sharp decrease, coincident with the terminal collapse of the circulation. In a second experiment, another animal was exposed to 6 per cent oxygen, and the cerebrospinal fluid pressure rose even in the face of a fall in the systemic arterial pressure. There was a maximal increase of 40 mm. of saline solution, which, however, was not sustained beyond half the duration of the exposure. The oxygen saturation of the blood fell from 15 to 4 volumes per cent. When 100 per cent oxygen was administered to the same animal, the increase in pressure showed a rapid subsidence to the preexperimental level, while at the same time the blood pressure showed some recovery. During a more rapidly conducted experiment with the same animal, a similar set of results was obtained. In the third experiment with the same animal, the rise in cerebrospinal fluid pressure tended to be maintained rather better, but showed a quick response on the administration of 100 per cent oxygen and again fell to normal. The author points out that although there was an initial rise in pressure, it was probably due to an increase in the cerebral arterial blood pressure, since the changes occurred before the content of oxygen in the blood was effectively lowered. However, a secondary and prolonged elevation was probably due to an accumulation of fluid from the cerebral capillaries under the influence of reduced oxygen saturation of the blood (average saturation, 41 per cent) and subsequent increased capillary permeability.

Maurer, in experiments on the effects of anoxia due to carbon monoxide poisoning, observed comparable increases in intracranial pressure. These increases neither had as rapid an onset nor subsided as rapidly on the giving of pure oxygen as did the increases in the experiments with low oxygen mixtures. This result may be attributed to the method employed, by which anoxia could actually be produced only by interference of the inert carbon monoxide with the oxygen-carrying power of the blood. This would account as well for the slower recovery from the anoxemia.

Nicholson,[7] in experiments with animals, allowed the cerebrospinal fluid to drip from a needle while the animals were subjected to breathing a low concentration of oxygen, namely, 6 per cent. Initially, in most instances, the low oxygen concentration caused a rapid and pronounced decrease in flow. With more prolonged administration the decrease was succeeded by an increase, the rate approaching, and in some cases exceeding, the preadministration level. In some experiments the initial decrease was preceded by a slight and brief increase. However, when low oxygen mixtures, of from 6 to 13 per cent, were given by constantly maintained artificial respiration, there was invariably a constant notable increase in the rate of flow of the cerebrospinal fluid. With prolonged administration this rate might drop slightly toward the end of the experiment, but in almost all experiments it was considerably above normal even then, that is, when the administration had lasted as long as forty minutes. Under these circumstances the administration of room air was followed by a quick return to the normal rate. In some cases the final rate was below normal. Although Nicholson

5. Haldane, J. S., and Priestley, J. G.: Respiration, ed. 2, New Haven, Conn., Yale University Press, 1935.

6. Maurer, F. W.: Effects of Anoxia Due to Carbon Dioxide and Low Oxygen on the Cerebrospinal Fluid Pressure, Am. J. Physiol. **133**:180, 1941.

7. Nicholson, H.: Effects of Low Alveolar Oxygen and High Alveolar Carbon Dioxide on the Rate of Flow of Cerebrospinal Fluid, Am. J. Physiol. **99**:570, 1932.

expressed the belief that these results indicated a change in the rate of production of the cerebrospinal fluid, it is also possible that an increase in the volume of the intracranial contents resulted in displacement of the cerebrospinal fluid in such a manner as to simulate increased production. Conceivably, such a change in relations might cause an increase in cerebrospinal fluid pressure in the manner described by White and associates.[2]

Secretion Pressure of Cerebrospinal Fluid.—The effect of anoxia on the permeability of the choroid plexus remains to be studied. By analogy with the concept that lack of oxygen increases capillary permeability, anoxia might easily be expected to effect an increased passage of fluid across this membrane system if the formation of the cerebrospinal fluid followed the principles of ultrafiltration. However, Flexner,[8] in considering the energy requirements for the formation of cerebrospinal fluid under normal conditions, pointed out that the hydrostatic pressure alone is inadequate to supply the necessary energy. The greater part of this energy must come from the cells of the choroid plexus, and hence it is concluded that the cerebrospinal fluid is not a dialysate in equilibrium with the blood plasma, nor can it be considered an ultrafiltrate of the blood plasma. Rather, it is a secretion representing work done by the cells of the choroid plexus.

The manner in which the anoxia due to increased altitude might affect the secretory power of the cells of the choroid plexus must have experimental investigation. Nicholson[7] concluded that anoxia could increase the production of cerebrospinal fluid. However, his experimental method does not allow the evidence he presented to be considered conclusive.

Rate of Absorption of Cerebrospinal Fluid.—In spite of the fact that in clinical medicine this factor appears to be of prime importance in some cases, it has not as yet been shown whether there is any alteration of the rate of absorption of cerebrospinal fluid under conditions of anoxia. Moreover, because anoxia causes increased permeability of vascular membrane, and because anoxia tends to increase cerebrospinal fluid pressure, it appears that factors associated with anoxia would hasten, rather than retard, the reabsorption of cerebrospinal fluid through the subarachnoid granulations. White and associate,[2] in considering the possible mechanisms which might serve to compensate for the swelling of the brain during anoxia and thereby result in absence of increase in the cerebrospinal fluid pressure, or a decrease after an initial increase, postulated the concurrence, in some cases, of an increase in the rate of absorption of the fluid through the normal channels, secondary to an increased hydrostatic pressure. This, they asserted, would be in accord with the Monro-Kellie hypothesis.

Elasticity of the Dura.—Weed, Flexner and Clark,[9] experimenting with small dogs, showed that the elasticity of the dura remained relatively constant under moderate variations of the cerebrospinal fluid pressure. In view of this constancy at ground level, one might question whether similar stability would not occur at a reduced atmospheric pressure. In order to influence cerebrospinal fluid pressure, however, any change would have to occur in disproportion to changes affecting the body as a whole. Since the body rapidly adjusts to reduced pressure, such selective changes seem unlikely. The relative avascularity of the dura as compared with adjacent cerebral structures makes it unlikely that anoxic vascular mechanisms would play an important role.

Intracranial Arterial Pressure.—The cerebral vascular reflexes are significant in maintaining the cerebral arterial pressure relatively constant even under conditions in which this might be altered as a result of systemic vascular changes. However, there are conditions in which increased arterial pressure, reflected intracranially, can influence cerebrospinal fluid pressure. Such a change is a common clinical observation in cases of hypertension.

Heymans and associates,[10] in experiments in which dogs were subjected to inhalation of nitrogen to produce acute anoxia, observed a rise in blood pressure in the intact animal. When the carotid sinuses had been denervated and the vagus nerves cut, inhalation of nitrogen caused a fall in the blood pressure.

Von Euler and Liljestrand,[11] working with dogs which were permitted to breathe an atmosphere containing 8.5 per cent oxygen, noted a slight increase in blood pressure, which was converted to a decrease after denervation of the carotid sinus.

8. Flexner, L. B.: The Chemistry and Nature of the Cerebrospinal Fluid, Physiol. Rev. **14**:161, 1934.

9. Weed, L. H.; Flexner, L. B., and Clark, J. H.: The Effect of Dislocation of Cerebrospinal Fluid upon Its Pressure, Am. J. Physiol. **100**:246, 1932.

10. Heymans, C.; Bouckaert, J. J., and Dautrebands, L.: Arch. internat. de pharmacodyn. et de thérap. **39**: 400, 1930; cited by Van Liere, E. J.: Anoxia: Its Effects on the Body, Chicago, University of Chicago Press, 1942.

11. von Euler, V. S., and Liljestrand, G.: Skandinav. Arch. f. Physiol. **74**:101, 1936; cited by Van Liere, E. J.: Anoxia: Its Effects on the Body, Chicago, University of Chicago Press, 1942.

White and associates [2] found no correlation between changes observed in the cerebrospinal fluid pressure and changes in systemic arterial pressure in cats in which swelling of the brain had been produced by anoxia.

As already mentioned, Maurer [6] obtained increases in intracranial pressure which, although they began coincident with slight elevations in blood pressure, continued to ascend even though the blood pressure subsequently fell to lower than the preadministration level.

Bergeret and Giordan [12] measured the arterial and cerebrospinal fluid pressures in dogs subjected to simulated high altitudes. The authors did not find any remarkable correspondence between the increases in the two pressures throughout the range of their investigation. Their observations will be discussed more fully in the next section.

Intracranial Venous Pressure.—Factors which influence intracranial venous pressure over any prolonged period must invariably involve obstruction of the venous drainage of the cranial cavity, for the free flow of venous blood would otherwise rapidly compensate for any intracranial change which would tend to compress venous channels and raise intracranial venous pressure.

Increased respiratory excursions, which are exhibited by persons brought to high altitudes without being given supplementary oxygen, would tend to facilitate the venous return to the thorax and thus reduce intracranial venous pressure and produce the well recognized phenomenon observed clinically, i.e., that deep breathing reduces cerebrospinal fluid pressure. An attempt to increase venous pressure by abdominal distention was recorded by Schaltenbrand.[13] In experiments with human subjects he injected air into the rectum until a pressure of 20 mm. of mercury had been reached. At that time the cerebrospinal fluid pressure, as measured manometrically through a lumbar puncture, showed an increase of 20 mm. of water, an insignificant change.

Bergeret and Giordan,[12] using dogs under anesthesia induced with chloralose (a compound of chloral hydrate and dextrose), recorded the femoral arterial pressure, the jugular venous pressure and the suboccipital cerebrospinal fluid pressure. They measured the cerebrospinal fluid pressure with an L-shaped manometer with a 4 mm. bore. When gas mixtures were administered, a tracheal cannula was used.

These authors described an experiment in which a slow ascent was made to 12,000 meters (approximately 39,000 feet) in seventeen and a half minutes, with plateaus at 8,000 and 10,000 meters (approximately 26,000 and 32,500 feet). While on the subject's reaching these plateaus slight increases were recorded in all pressures, on his attaining 12,000 meters, the arterial pressure had risen from 10 to 30 mm. of mercury; the venous pressure had remained relatively the same, and the cerebrospinal pressure had shown an increase of 25 to 40 mm. of water. While the subject remained at 12,000 meters, the arterial pressure showed a tendency to drop, about 80 mm. of mercury in all being lost; the venous pressure increased 150 mm. of water, and the cerebrospinal fluid pressure continued to rise, showing a total increase of 150 mm. of water over its initial value. After a rapid descent to ground level, the venous pressure rapidly decreased to normal; the arterial pressure returned to its normal value after a brief peak, and the cerebrospinal fluid pressure slowly descended to the initial value over a period of twenty-five minutes.

The authors described another experiment in which a rapid ascent was made to 12,000 meters. The arterial pressure initially rose approximately 80 mm. of mercury; the venous pressure and the cerebrospinal fluid pressure showed a slower tendency to increase, but toward the end of the time that the animal was held at 12,000 meters the cerebrospinal fluid pressure had increased about 200 mm. of water and the venous pressure had risen 190 mm. of water. Both these pressures, as well as the arterial pressure, fell off shortly before the descent began. However, the pronounced rise which was shown in both the venous and the cerebrospinal fluid pressure occurred synchronously with a fall in arterial pressure of about 70 mm. of mercury below its initial value.

In another ascent to 12,000 meters, it was found that during the ascent, which consumed nine minutes, the cerebrospinal fluid pressure rose from 280 to 440 mm. of water, while the arterial pressure rose about 140 mm. of mercury. During this time there had been no increase in venous pressure. When the altitude attained 12,000 meters, there was a sharp increase in venous pressure, amounting approximately to 120 mm. of water, at which time the cerebrospinal fluid pressure continued to rise an additional 240 mm. of water, i. e., approximately 400

12. Bergeret, P., and Giordan, P.: Influence of Atmospheric Depression on the Cerebrospinal Fluid Pressure, J. de physiol. et de path. gén. **36**:1050, 1938.

13. Schaltenbrand, G.: Atmospheric Pressure, Circulation, Respiration and Cerebrospinal Fluid Pressure: II. Oxygen Pressure and Cerebrospinal Fluid Pressure, Acta aerophysiol. (no. 1) **1**:65, 1933.

mm. of water in all. At this point oxygen was introduced. The arterial and jugular venous pressures returned to normal. The cerebrospinal fluid pressure dropped 270 mm. of water. Administration of oxygen was stopped, and the animal was allowed to remain at 12,000 meters. The cerebrospinal fluid pressure returned to its former high value. The jugular venous pressure was also increased. Coincident with the maximum increases in both these pressures, there was a notable fall in arterial pressure. A descent was then carried out rapidly. The arterial pressure recovered; the jugular venous pressure returned rapidly to normal, and the cerebrospinal fluid pressure returned over a period of about fourteen minutes.

Bergeret and Giordan also produced decompression by subjecting an animal to an altitude of 8,000 meters after denervation of the carotid sinus. The arterial pressure showed no rise, but, rather a gradual fall. The jugular venous pressure showed only a minor increase. The cerebrospinal fluid pressure showed an increase of 50 mm. of water.

The authors concluded that in a dog anesthetized with chloralose decompression caused an increase in cerebrospinal fluid pressure which could become pronounced if the decompression was sufficiently prolonged. They concluded that the variations were essentially passive and that they were dependent on alterations in the general circulation, especially changes in the venous pressure. Bergeret and Giordan expressed the opinion that the arterial pressure had a role in the initial increases in cerebrospinal fluid pressure. The increase in cerebrospinal fluid pressure and, likewise, in venous pressure ceased when the animal breathed oxygen, even though he continued at the same level of decompression. The authors did not state whether they observed any notable increase in abdominal distention coincident with increases in venous pressure. It is unlikely that these changes in venous pressure would not be transmitted to the cranial cavity and influence to some extent the rise in cerebrospinal fluid pressure.

The changes in arterial pressure which were seen at the time of the initial rise may also have played a part, but the most extreme increases in cerebrospinal fluid pressure occurred when the arterial pressure had shown a significant fall. However, the authors failed to mention any explanation for their changes in cerebrospinal fluid pressure which would consider swelling of the brain tissue due to anoxia. In view of the observations of other investigators, this factor cannot be neglected. Moreover, the slow return of the cerebrospinal fluid pressure to normal is consistent with this view.

Their observation that administration of oxygen to an animal while it remained at the same degree of decompression would reverse the changes in pressure of the cerebrospinal fluid is important evidence to support the contention that anoxia is the basis of changes in the cerebrospinal fluid pressure at high altitudes. However, in the absence of data on analysis of gases in the chamber atmosphere, as well as in the blood, one cannot be sure that the mixture of gases breathed by the animals at pressures corresponding to various altitudes was exactly comparable to atmospheric air as it occurs at those altitudes. Inadequate ventilation of a decompression chamber can produce anoxia, since oxygen is used up by the animal more rapidly than it can be replaced in the chamber.

Bergeret and Giordan also described the use of a manometer with a bore of 4 mm. It was shown by Weed, Flexner and Clark[9] that the bore of a manometer can influence the recorded pressure of the cerebrospinal fluid pressure because of the factor of dislocation of fluid. In one experiment, they described a difference of over 50 mm. of water between the pressure measured by a manometer of 1 mm. bore and that measured by an instrument of 4 mm. bore.

ANALYSIS OF STUDIES ON CEREBROSPINAL FLUID PRESSURE AT HIGH ALTITUDE

Walsh[14] described the case of a patient who had had a craniotomy in the right temporal area and who was subjected to simulated high altitudes in a decompression chamber while breathing a high concentration of oxygen. The highest altitude reached was 28,000 feet (8,500 meters). It was observed that the scalp overlying the defect in the skull had risen 1 cm. Although in this case the scalp was recorded as having moved outward, consideration of such a factor as the subject's emotional adjustment to being in a decompression chamber is not mentioned. As will be noted later, emotional responses in experiments with simulated decompression can produce slight increases in pressure. Moreover, since plethysmographic technic was not employed, one cannot say what change in tension occurred which might have caused this slight bulging. Thus, the results of this experiment are entirely qualitative and open to question.

14. Walsh, M. N.: Changes in Intracranial Volume on Ascent to High Altitudes and Descent As in Diving, Proc. Staff Meet., Mayo Clin. **16**:220, 1941.

Walsh and Boothby[15] performed lumbar punctures on 3 subjects and subjected them to decompression at an altitude of 30,000 feet (approximately 9,000 meters). They noted bubbles of gas, presumably nitrogen, first appearing in the water column of the manometer at an altitude of between 10,000 and 12,000 feet (3,000 and 3,600 meters). There was an increase in pressure of 30 mm. of water during this ascent to 30,000 feet (9,000 meters). One cannot feel that, under the conditions in which human cerebrospinal fluid pressure is recorded, a difference in pressure of 30 mm. of water is significant.

Armstrong,[16] using goats with puncture needles placed in the cisterna magna and with fitted oxygen masks subjected the animals to decompression at an altitude of 50,000 feet (about 15,000 meters). At 18,000 feet (5,500 meters) the cerebrospinal fluid pressure began to rise, the increase being synchronous with the appearance of gas bubbles in the manometer. The pressure continued to rise with decompression to 50,000 feet (15,000 meters). The author stated that the pressure was never more than a few inches (1 inch = 25 mm.) of water over normal. Armstrong also claimed to have repeated similar experiments with human subjects and to have obtained similar results. It is significant that in this work, although anoxia was controlled, goats were used as subjects and the factor of abdominal distention seems not to have been considered. Also, the presence of bubbles in the manometer would appear to complicate accurate pressure readings. As the author stated, the increase in pressure was intermittently relieved as bubbles passed through the manometer and out into the atmosphere.

The same author experimented with animals the brains of which had been exposed through trephine holes. He did not note what animals were used or whether the factor of anoxia was controlled. He stated, however, that there was progressive bulging of the brain, which began to herniate through the opening at 30,000 feet (9,000 meters). He noted that this condition was relieved on descent. One may presume that in such a case if the skull were intact increased intracranial pressure would have occurred. However, in the absence of knowledge as to how well the experiment was controlled, one cannot state which of the factors of change in altitude was responsible for the results.

Schaltenbrand[17] subjecting human beings under sedation induced with phenobarbital and dogs under chloralose anesthesia to simulated high altitudes in a decompression chamber, found that the cerebrospinal fluid pressure began to increase at about 3,000 meters (10,000 feet) in man and at a somewhat higher altitude, 16,000 feet (5,000 meters), in dogs. He recorded an increase of 190 mm. of water in a human subject at 15,000 feet (4,600 meters). Similarly in dogs there was an increase of 130 mm. of water at 26,000 feet (8,000 meters). As a control observation, it was noted that the phychologic effect of sham decompression caused an increase in cerebrospinal fluid pressure of 40 mm. of water.

In investigating the importance of anoxia, Schaltenbrand observed that when an altitude of about 17,000 feet (5,200 meters) had been reached, and the cerebrospinal fluid pressure had risen 150 mm. of water, the administration of oxygen reduced the pressure to normal values. Similar results were noted with other patients. In another experiment, in which the altitude was slightly greater, 20,000 feet (6,150 meters), administration of oxygen reduced only in part the elevation of cerebrospinal fluid pressure, i.e., a decrease of 100 mm. from an increase of 270 mm. of water. However, with patients subjected to decompression in an atmosphere containing 40 to 45 per cent oxygen at an altitude of 21,000 feet (6,460 meters) the cerebrospinal fluid pressure increased by only 40 mm. of water, an insignificant amount. At the same altitude, the same patient, when breathing air, had an increase in cerebrospinal fluid pressure of 200 mm. of water.

To test the factor of abdominal distention in production of increases in cerebrospinal fluid pressure, air was injected through the rectum into the bowel until the pressure had reached a level of 20 mm. of mercury. This resulted in an insignificant increase of cerebrospinal fluid pressure, namely, 20 mm. of water.

Other experiments, in which dogs anesthetized with chloralose were employed, failed to indicate any correlation between changes in systemic arterial pressure and the cerebrospinal fluid pressure under conditions of increased altitude. Although

15. Walsh, M. N., and Boothby, W. M.: Demonstration of Air Bubbles in Spinal Fluid Under Lowered Atmospheric Pressures Produced in a Low Pressure Chamber, Proc. Staff Meet., Mayo Clin. **16**:225, 1941; correction, ibid. **16**:304, 1941.

16. Armstrong, H. G.: Aviation Medicine, Baltimore, Williams & Wilkins Company, 1939.

17. Schaltenbrand, G.: Atmospheric Pressure, Circulation, Respiration and Cerebrospinal Fluid Pressure: Atmospheric and Cerebrospinal Fluid Pressure, Acta aerophysiol. (no. 1) **1**:61, 1933; Atmospheric Pressure, Circulation, Respiration and Cerebrospinal Fluid Pressure: III. Relation of Cerebrospinal Fluid Pressure and Blood Pressure and Respiration, ibid. (no. 2) **1**:41, 1933.

the changes in blood pressure were great in some cases, they were often variable and often opposite in direction to the progressive and constant increases in cerebrospinal fluid pressure which he obtained in these animals.

From his studies, Schaltenbrand concluded that diminution of atmospheric pressure with increased altitude beyond a critical point—for man 10,000 feet (3,000 meters) and for anesthetized dogs 20,000 feet (6,100 meters)—caused an increase in cerebrospinal fluid pressure. He concluded that these changes were chiefly the effects of anoxia and that they could be prevented or abolished by the administration of oxygen. He stated the opinion that arterial pressure did not play a part in the increases in cerebrospinal fluid pressure which he noted.

Schaltenbrand, in his studies on human subjects, employed patients with mental disorders. Sedation was induced with phenobarbital. To what extent such persons would react to the surroundings of a decompression chamber is difficult to state. Conceivably, some of the larger increases in cerebrospinal fluid pressure which the author observed may have been related to the factor of psychologic stress. This may explain the fact that he could not in all cases inhibit increases in cerebrospinal fluid pressure at higher altitudes by means of administration of oxygen. He did not attempt to determine the exact degree of anoxia produced by given amounts of oxygen in the blood.

COMMENT

In the reports that have just been analyzed, several authors [18] claimed to show increases in intracranial pressure under conditions applying at increased altitudes. Several of these reports failed to supply all the details necessary for a complete assessment of the results presented. For the most part, the changes at reasonable altitudes were not great. In reports in which necessary details were furnished, the observed changes in intracranial pressure could be related to changes in the physical environment not strictly the result of altitude itself. These changes appear capable of experimental isolation, in an attempt to discover what effects, if any, they alone can produce.

It may be safely concluded that the body rapidly adjusts to changes in atmospheric pressure due to increase in altitude and that alterations in the cerebrospinal fluid pressure on the basis of reduced atmospheric fluid pressure due to increased altitude do not occur. In carnivorous animals or in man,[13] when distention of the bowel from contained gas under conditions of decreased pressure due to high altitudes is not great, increase in intracranial pressure from decreased atmospheric pressure will in all probability not be very great, certainly not great enough to be of clinical significance.

This leaves the other important factor associated with altitude, namely, the reduced oxygen tension, as a possible influence. Certainly, at altitudes which would be capable of causing anoxia of moderately severe degree there might be increases in intracranial pressure. At higher altitudes there would be more danger of deterioration, and hence the pressure might not tend to rise appreciably. In fact, it might fall. Thus, in any discussion of the effect of altitude on the intracranial pressure, it must always be borne in mind that the factor of anoxia must be controlled. When this control prevents the occurrence of anoxia, the intracranial pressure may be expected to show no appreciable increase.[19] If anoxia of moderately severe degree should occur, the intracranial pressure may be expected to increase, although experimental evidence would lead one to believe that this is not an invariable result.[3] Further, regardless of the concentration of oxygen in the gas mixture being breathed, due consideration must be given to estimations of the oxygen content of the arterial blood, since Michelson and Thompson [4] found that when gas mixtures were breathed, the oxygen saturation of the blood in different persons tended to vary over an appreciable range. The reason for this difference cannot be attributed to any single factor but probably is related to differences in degree of activity and metabolism. One might expect that such differences would have a bearing on the degree of increase of intracranial pressure which might be obtained among different persons under similar conditions of reduced oxygen tension.[4] It was pointed out by White and associates [2] that the onset of cerebral edema coincides with a decrease in the oxygen saturation of the blood to about 85 per cent of the normal value. This decrease would correspond to that obtained at an altitude of about 11,000 feet (3,380 meters) when supplementary oxygen is not supplied. Possibly this degree of anoxia can in many instances be sustained without any remarkable increase in intracranial pressure. However, further investigation is required to prove this point.

Bergeret and Giordan [12] describe the relief of increased venous pressure by the administration

18. Michelson and Thompson.[4] Nicholson.[7] Bergeret and Giordan.[12] Schaltenbrand.[18] Walsh.[14] Walsh and Boothby.[15] Armstrong.[16] Schaltenbrand.[17]

19. Bergeret and Giordan.[12] Walsh and Boothby.[15] Schaltenbrand.[17]

of oxygen while the animals remained at a decompression level. However, the full increment in intracranial pressure which had been produced by anoxia was not immediately relieved. Thus, it appears that the production of increased intracranial pressure was due to some factor in addition to increased venous pressure. Whether this factor was in itself secondary to increased venous pressure would be difficult to state. Since the pressure returned slowly to normal when the venous pressure was relieved, and since other investigators [2] have shown that cerebral swelling accompanies anoxia, it may be concluded that cerebral swelling, either due to anoxia itself or secondary to increased venous pressure during anoxia, was responsible, at least in part, for the changes which these authors described.

The fact that increased intracranial pressure does not invariably follow the onset of cerebral edema was pointed out by White and associates [2] in their experiments on cats. They stated the opinion that the factor of increased absorption or of decreased production of fluid is important in compensating for the increased size of the cerebral tissues and affords a compensatory mechanism against the development of increased intracranial pressure. If this is true, the relations postulated in the Monro-Kellie hypothesis continue to apply. Also, Bergeret and Giordan by careful measurement of the volumes of the brain and skull and by comparison of the ratios of the volumes with similar ratios obtained for brains of subjects in whom anoxia had never been produced, found a qualitative increase in the volume of the cerebral tissue. Since in most instances the intracranial pressure did not rise, or, if it rose, it subsequently fell, one may consider that changes in the rate of production or of absorption of the cerebrospinal fluid may have been responsible for this tendency of the pressure to remain stable.

CONCLUSION

There is no evidence that reduced atmospheric pressure itself has any influence on the intracranial pressure.

The expansion of gas in the bowel at reduced atmospheric pressures may cause increases in intracranial pressure secondary to a rise in venous pressure.

Anoxoia due to breathing air at reduced atmospheric pressure may cause increased intracranial pressure, as a result of the production of cerebral edema.

The increase in arterial blood pressure due to the anoxia of high altitudes may be a possible factor in the initial increases of intracranial pressure.

Changes in the rate of production or of absorption of cerebrospinal fluid under conditions of anoxia may serve to compensate against increases in intracranial pressure produced by the mechanisms listed in the preceding three paragraphs.

Increases in intracranial pressure observed in studies on man and on experimental animals subjected to high altitudes have not been of such magnitude as to give them great clinical significance.

For the proper investigation of intracranial pressure at various altitudes, the following factors should be studied quantitatively: (*a*) venous pressure; (*b*) presence of abdominal distention; (*c*) oxygen tension in the gas mixture breathed; (*d*) oxygen saturation of the blood; (*e*) cerebrospinal fluid pressure, determined with a manometer of not more than 1 mm. bore; (*f*) in animal experiments, amount of cerebral edema due to anoxia; (*g*) chronicity of the anoxia, and (*h*) in experiments on man, emotional response and bodily relaxation, and, in experimental animals, relaxation as influenced by the depth of anesthesia.

Experimental evidence as it appears in the literature indicates that significant increases in the intracranial pressure will not occur in the sick or wounded during transportation by air provided anoxia is not permitted to develop.

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DETECTION OF THE LATENT BABINSKI SIGN WITH SCOPOLAMINE

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In 1903 it was pointed out by Volkmann[1] that normal plantar responses became extensor after the administration of scopolamine, and after this report there appeared in the literature sporadic papers reaffirming this observation. Some authors stated the opinion that pyramidal signs were not evoked by scopolamine in the absence of damage to the pyramidal tract and suggested that scopolamine be used clinically as a test to bring out latent pyramidal signs. From a review of the literature, it appears that this test has never been widely used or generally accepted as having any clinical value. We have found reference to its use in only one of the standard neurologic textbooks in English, and that in a footnote.[2] Yet the need of a simple test to facilitate the detection of the Babinski sign is evident to any one who has occasion to evaluate the varied types of plantar responses encountered in a routine neurologic examination. With this in mind, it seems that the older views concerning the employment of scopolamine for this purpose need reviewing and the usefulness of the test requires evaluation.

REVIEW OF LITERATURE

Volkmann[1] (1903) noted the appearance of the Babinski sign in patients undergoing major surgical procedures while under morphine-scopolamine anesthesia. Scopolamine, 2.7 mg., and morphine, 35 mg., were given in a period of a little over six hours. With this dose, consciousness was lost; reflexes were lost or became hypoactive; muscle tone was lowered and sometimes disappeared, and the Babinski sign was nearly always observed.

Link[3] (1905), using scopolamine as a sedative, or for the induction of narcosis in mentally deranged patients, found the Babinski sign present in 31 of 36 patients. Doses between 0.3 and 2 mg. were used, and the smallest dose which produced this sign was 0.4 mg. The drug was invariably given with morphine, but independent observations indicated that the reflex change in question was due solely to scopolamine. In contrast to Volkmann's observation, Link noted an increase of deep reflexes and frequent ankle clonus, in addition to lowered muscle tone and extensor plantar responses. This author expressed the belief that the elicitation of extensor plantar responses was dependent on the functional cortical depression brought on by scopolamine and that it was similar to the appearance of this sign in deep physiologic sleep and in epileptic coma.

Hahn[4] (1911) studied many cases of chronic alcoholism and convinced himself that the presence of extensor plantar responses was always due to secondary organic lesions. He mentioned that in 1 case uncertain responses were made definitely extensor after an injection of scopolamine.

Rosenfeld[5] (1921) obtained extensor plantar responses in excited, physically exhausted patients with mental disorders by administration of 0.5 to 1 mg. of scopolamine, given for control of motor excitation. Barbital, chloroform and sodium barbital did not produce this response in his patients. In some patients questionable plantar responses became definitely extensor after the use of scopolamine. Rosenfeld was unable to produce a similar response in patients with purely extrapyramidal lesions and stated the opinion that a negative result in the scopolamine test is of diagnostic value when differentiation of lesions of the pyramidal and of the extrapyramidal tract is required.

Lemos[6] (1923), using scopolamine in a dose of 1.2 mg. in treating patients with postencephalitic parkinsonism, noted the appearance of pyramidal signs in the form of hyperreflexia, ankle

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1. Volkmann, G.: Beiträge zur Morphium-Skopolamin-Narkose, Deutsche med. Wchnschr. **29**:967, 1903.

2. Monrad-Krohn, G. H.: The Clinical Examination of the Nervous System, ed. 5, New York, Paul B. Hoeber, Inc., 1930, p. 100.

3. Link, R.: Ueber den Babinski'schen Reflex, Neurol. Centralbl. **24**:630, 1905.

4. Hahn, R.: Babinski'sches Zeichen und verwandte Erscheinungen unter Einwirkung von Hyoszin und Alcohol, Neurol. Centralbl. **30**:185, 1911.

5. Rosenfeld, M.: Ueber Scopolaminwirkungen am Nervensystem, München. med. Wchnschr. **68**:971, 1921.

6. Lemos, M.: Action de la scopolamine sur le clonus et la reflectivité en général dans un cas de syndrome Parkinsonien postencéphalitique prolongé, Rev. neurol. **2**:424, 1923.

clonus and extensor plantar responses. He suggested that this drug might be used to reveal clinically latent involvement of the pyramidal system.

Elliot and Walshe[7] (1925), in discussing the appearance of the Babinski sign in various toxic states, mentioned the use of scopolamine in unmasking a doubtful or hidden extensor response. In several patients with parkinsonism and in 1 patient with a tumor of the frontal lobe, this effect was obtained after injections of 1/100 to 1/150 grain (0.6 to 0.4 mg.) of scopolamine.

Zador[8] (1927) obtained extensor plantar responses in 3 of 4 patients with postencephalitic parkinsonism by administration of doses of 1.2 to 1.5 mg. of scopolamine; he also reported the appearance of this sign in normal persons given scopolamine in doses over 1 mg.

The most extensive work on this subject was that done by Delmas-Marsalet.[9] Working principally with patients having parkinsonism due to encephalitis, this author observed the appearance of pyramidal signs after injections of 2.5 mg. of scopolamine. Without citing his experimental evidence or the dosage used, he stated that scopolamine will not produce these signs in normal persons. In his monograph on the elementary reflexes of posture, Delmas-Marsalet[9b] discussed the antagonism between these reflexes and the appearance of pyramidal signs. He expressed the opinion that scopolamine provokes the appearance of pyramidal signs by abolishing what he called the elementary postural reflexes. He reported that the pyramidal signs were in evidence within thirty-five to sixty minutes after the subcutaneous injection of 1.25 mg. of scopolamine. He also suggested that the scopolamine test be used to unmask latent pyramidal signs in cases of postencephalitic parkinsonism.

METHOD

In an attempt to study critically the foregoing observations and to determine the clinical applicability of the scopolamine test, a series of control subjects and of patients with various neurologic disorders were given scopolamine hydrobromide. The drug was administered subcutaneously in doses of 0.3 or 0.4 mg. These doses produced varying degrees of giddiness, somnolence, dryness of the mouth, flushing, tachycardia and paralysis of accommodation. In several patients given 0.6 mg. of the alkaloid these reactions were severe, and in subsequent tests the dose was reduced to the former level. A patient, aged 72, with progressive muscular atrophy became considerably disoriented and was mentally confused for several hours after receiving 0.4 mg. of the drug. However, in no instance was the reaction so severe as to contraindicate the use of the 0.3 or 0.4 mg. dose. All the toxic symptoms disappeared more rapidly when 10 mg. of amphetamine sulfate was given by mouth at the end of the period of observation. The data we obtained from tests in which we gave amphetamine simultaneously with the scopolamine were insufficient to ascertain whether the results with this method would differ from those noted with scopolamine alone.

The patients were examined at intervals of thirty minutes, forty-five minutes and one, one and a half and two hours after receiving the scopolamine. In none of them were there any detectable changes in muscle tonus or in the state of the tendon or cutaneous reflexes, except for the alteration in the plantar response, to be described later. The change in the plantar response appeared to be independent of the degree of severity of the toxic symptoms produced by the scopolamine. In all cases in which the plantar response became extensor in type this change occurred by the end of the first hour. In the light of these observations, the technic can be simplified by testing for the Babinski sign just before the administration of scopolamine and again one hour afterward.

RESULTS

A series of 47 patients without damage to the pyramidal tract were tested with scopolamine, and in no instance could the normal flexor plantar response be reversed or any other abnormal signs elicited. This series included 24 patients admitted to the hospital for treatment of various medical conditions, 12 patients with miscellaneous neurologic disorders in whom no damage to the pyramidal tract was evident and no latent pyramidal signs were suspected, and 11 patients showing localized muscular atrophy or fibrillations or with reflex changes sufficiently pronounced to suggest the possibility of disease of the pyramidal tract. These 11 patients were thoroughly studied, and in the final analysis it was concluded that their disorders were not early progressive muscular atrophy, tumor of the cord, syringomyelia or any of the conditions most commonly considered as diagnostic possibilities.

Another series of 40 patients with evidence of damage to the pyramidal tract but with absence of the Babinski response, or with an equivocal one, were tested with scopolamine. In 23 of these patients the plantar response became unquestionably extensor. This change occurred in 9 of 11 patients with multiple sclerosis; in 5 of 10 patients with progressive muscular

7. Elliot, T. R., and Walshe, F. M. R.: The Babinski or Extensor Form of Plantar Response in Toxic States Apart from Organic Disease of the Pyramidal Tract System, Lancet **1**:65, 1925.

8. Zador, J.: Ueber die Beeinflussbarkeit und Pathogenese des Babinskischen Reflexes, Monatschr. f. Psychiat. u. Neurol. **64**:336, 1927.

9. Delmas-Marsalet, P.: (*a*) La scopolamine, réactif des lesions pyramidales latentes de certains parkinsoniens: Mecanisme de son action, Compt. rend. Soc. de biol. **94**:1153, 1926; (*b*) Lés réflexes de posture élémentaires, Paris, Masson & Cie, 1927.

atrophy of the flaccid type; in 3 of 10 patients with cerebral lesions, including tumors, vascular lesions and post-traumatic epilepsy, and in 2 of 5 patients with pernicious anemia showing only signs referable to the posterior column. In a patient with tuberculosis of the spine, in another with a tumor of the cervical portion of the cord, in another with a herniated cervical intervertebral disk and in another with a condition the diagnosis of which lay between syringomyelia and an intramedullary tumor of the cord the initial equivocal plantar responses became definitely extensor after administration of scopolamine. In our opinion, in none of the aforementioned patients did a latent Babinski sign appear in the absence of damage to the pyramidal tract.

SUMMARY

As an aid in neurologic diagnosis, a test using scopolamine to facilitate the detection of the latent Babinski sign was employed. In none of 47 control patients did an extensor plantar response become evident after the test. In 23 of 40 patients with suspected or known damage to the pyramidal tract, but with normal or atypical plantar responses, a latent extensor response was brought out with this test. From these observations we conclude that the subcutaneous administration of scopolamine in the proper dose is a reliable and practical clinical test for the detection of a latent extensor plantar response, serving thus to unmask concealed damage to the pyramidal tract.

Duke Hospital.

BASILAR IMPRESSION RESEMBLING CEREBELLAR TUMOR

REPORT OF A CASE

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Since 1939, when Chamberlain [1] focused attention on the pathologic complex of basilar impression and its concomitant neurologic manifestations, a dozen or more American observers have reported a total of 19 verified cases in which the condition masqueraded as a variety of syndromes. The literature on the subject has been reviewed by several of these authors, who credited Rokitansky with having first mentioned the deformity, in 1844. Homén,[2] in 1901, and Schüller,[3] in 1911, correlated the neurologic observations, and the latter admirably described the roentgenographic changes in patients with the anomaly.

The condition is generally accepted as representing, basically, a developmental anomaly of the craniovertebral boundary, the most striking feature being an invagination of the basiocciput by an upward projection of elements of the atlas and axis. When they are viewed from inside the cranial cavity, the foramen magnum is stenosed and the clivus forms a convex band, which may be elevated as high as the sella turcica. The basiocciput is usually thinned and the condyloid process often rudimentary. The atlas is commonly fused with the occiput. These skeletal malformations are often associated with other bony deformities, involving most commonly other cervical vertebrae. Chamberlain denied the possible etiologic factor of rickets, osteomalacia or other systemic skeletal diseases in which the skull is softened. Aside from a primary basilar impression, however, the condition may develop secondary to certain osteolytic diseases. Ray,[4] reported a case of osteitis deformans and another of osteogenesis imperfecta in both of which a basilar invagination developed, with the subsequent neurologic changes. The term "basilar impression" should, therefore, be limited to those anomalies thought to be primary invaginations. The term "platybasia," though made popular by Chamberlain, is a misnomer, deplored by Walsh and Craig,[5] who pointed out that anthropologically platybasia denotes an abnormal obtuseness of the sphenoid angle. The sphenoid angle is the angle between the line from the foremost point of the foramen magnum to the center of the sella turcica and the line from this point to the roof of the nose. The angle varies normally from 115 to 150 degrees. Basilar impression may or may not be associated.

It is popularly believed that this congenital malformation of the craniovertebral boundary eventually leads to encroachment of bone on the adjacent neural structures, notably the medulla, cerebellum, cranial nerves and upper cervical portion of the cord. Lichtenstein [6] pointed out that the reverse of this cause-effect relationship is seen in the Arnold-Chiari syndrome.

It is difficult to explain why in cases of primary basilar impression symptoms appear as late as the second, third or fourth decade of life. Light's explanation, cited by Chamberlain,[1] can apply only to cases in which neurologic symptoms develop near puberty. He suggested that

1. Chamberlain, W. E.: Basilar Impression (Platybasia): A Bizarre Developmental Anomaly of the Occipital Bone and Upper Cervical Spine with Striking and Misleading Neurological Manifestations, Yale J Biol. & Med. **11**:487-496, 1939.

2. Homén, E. A.: Zur Kenntnis der rachitischen Deformationen der Schädelbasis, Deutsche Ztschr. f. Nervenh. **20**:3-15, 1901.

3. Schüller, A.: Zur Röntgen-Diagnose der basaler Impression des Schadels, Wien.med.Wchnschr. **61**:2593-2599, 1911.

4. Ray, B. S.: Platybasia with Involvement of Central Nervous System, Ann. Surg. **116**:231-250, 1942.

5. Walsh, M. N.; Camp, J. D., and Craig, W. M.: Basilar Invagination of Skull (So-Called Platybasia): Case with Operation, Proc. Staff Meet., Mayo Clin. **16**:449-452, 1941.

6. Lichtenstein, B. W.: Distant Neuroanatomic Complications of Spina Bifida (Spinal Dysraphism), Arch. Neurol. & Psychiat. **47**:195-214 (Feb.) 1942.

the spinal cord has already attained its normal adult length at a time, just before puberty, when the skeletal system takes a normal and characteristic spurt in growth, with the production of traction on the cord. Gustafson and Oldberg [7] offered the further suggestion that chronic and mild impairment of the neurodynamics due to mechanical compression is compensated for a while. Other authors presented the corollary that the more robust nerve tissue of youth loses its tolerance to such mechanical pressure concomitantly with the aging process.

Undoubtedly, several underlying factors are concerned in the ultimate complex. This becomes apparent when one reviews the 19 cases previously referred to. In many cases of basilar impression neurologic signs are known never to have developed. In the cases in which signs do appear, the symptom complex may be confused with a number of neurologic diseases. In Chamberlain's [1] 4 cases the disease simulated syringomyelia, disseminated sclerosis and varieties of spastic paralysis. Gustafson and Oldberg [7] reported 3 cases associated with syringomyelia and 1 with hydrocephalus. Lichtenstein,[8] in 1943, presented further evidence that syringomyelia may develop from degeneration of central portions of the cervical portion of the cord due to bony impingement on the neuraxis itself or on overlying vessels, with consequent ischemia of the cord. Such cases may of course represent only coincidental myelodysplasia or true syringomyelia. List's [9] 4 cases represented syndromes of compression of the cerebellum, bulb and cervical portion of the cord. Craig, Walsh and Camp [10] reported 3 cases with similar manifestations. In Ray's [4] 2 cases the disease masqueraded as multiple sclerosis and syringomyelia respectively, while Stephens [11] reported 2 cases of basilar impression with manifestations of a bulbar type of amyotrophic lateral sclerosis, in 1 of which great improvement was effected by occipitocervical decompression.

The case here presented is unique in that the signs and symptoms were strictly limited to cerebellar manifestations, and if roentgenograms of the skull had been ignored a diagnosis of cerebellar tumor would have been entirely plausible.

The diagnosis of basilar impression rests on roentgenographic evidence. In lateral views of the skull one sees cervical spine elements lying above the so-called Chamberlain line, i. e., a line drawn from the posterior border of the foramen magnum to the roof of the mouth. There is commonly a fusion of the first or the first and second cervical vertebrae with the occiput. Chamberlain also emphasized the importance of the open mouth roentgenographic technic, in which one obtains through the pharynx an obstructed anteroposterior view of the craniovertebral articulations. Jostes [12] reported that with the use of the laminograph in such anteroposterior views the overlying maxilla can be eliminated and the plane of the occipital condyles and the atlanto-occipital joints brought out in sharp focus, the results exceeding the limitations of the routine, open mouth method.

It has frequently been demonstrated that surgical decompression of the involved neural axis may if performed early in the course of the illness provide arrest of further damage.

REPORT OF CASE

History.—A. F., a white man aged 55, was admitted on March 10, 1944, with the following history: He had enjoyed good health prior to October 1941, when he first noticed instability of gait, especially when fatigued. This became progressively more apparent during the next ten months, at which time his gait was constantly ataxic. Seven years prior to the time of admission he had sustained a blow to the top of the head in an automobile accident. He was unconscious for one hour but was said to have no injury other than a scalp wound. He stated he had always been able to tolerate large amounts of liquor, but two years ago he noticed that a single drink of whisky would make him "very drunk." For the past one and one-half years the patient had had frequently recurring dull headaches (of vague location) and spells of dizziness.

First Hospitalization.—He was seen elsewhere in August 1943, and a diagnosis of platybasia was made on the basis of the roentgenographic evidence and cerebellar symptoms. The fundi were said to be normal at this examination. The patient's condition failed to improve under a regimen of dehydration, and an exploration of the posterior fossa was made. The preoperative diagnosis was confirmed. The cerebellar tonsils were displaced downward under the lamina of the atlas,

7. Gustafson, W. A., and Oldberg, E.: Neurologic Significance of Platybasia, Arch. Neurol. & Psychiat. **44**:1184-1198 (Dec.) 1940.

8. Lichtenstein, B. W.: Cervical Syringomyelia and Syringomyelia-Like States Associated with Arnold-Chiari Deformity and Platybasia, Arch. Neurol. & Psychiat. **49**:881-894 (June) 1943.

9. List, C. F.: Neurologic Syndromes Accompanying Developmental Anomalies of Occipital Bone, Axis and Atlas, Arch. Neurol. & Psychiat. **45**:577-616 (April) 1941.

10. Craig, W. M.; Walsh, M. N., and Camp, J. D.: Basilar Invagination of Skull, So-Called Platybasia: Three Cases in Which Operation Was Done, Surg., Gynec. & Obst. **74**:751-754, 1942.

11. Stephens, R. H.: Platybasia: A Report of a Case Treated Surgically with Improvement, Surgery **12**:943-951, 1942.

12. Jostes, F. A.: Neck Pain: Laminagraph as Aid to Diagnosis of Atlanto-Occipital Lesions, J. A. M. A. **118**:353-359 (Jan. 31) 1942.

and beneath this there was a definite bulge of the medulla. A wide suboccipital decompression was accomplished and a laminectomy of the first cervical vertebra made. The dura was not closed, but the defect was covered with animal membrane. After operation the patient enjoyed temporary improvement and was discharged on August 25.

Fig. 1.—Lateral view of the skull, revealing an old postoperative defect in the basiocciput and persistence of cervical elements above Chamberlain's line, i. e., a line drawn from the posterior border of the foramen magnum to the bony palate.

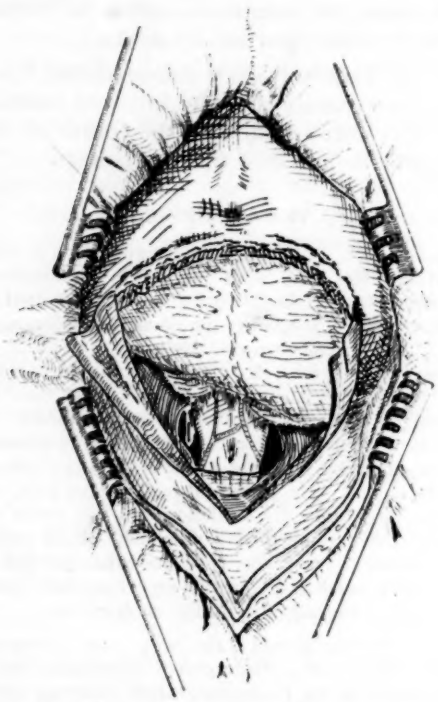

Fig. 2.—Initial view of the posterior fossa on reflection of the dura, showing a dense sheet of arachnoid constricting the atrophied cerebellum.

Postoperative Course.—Subsequently, however, there was progressive recurrence of the cerebellar symptoms. Headaches became more frequent and severe. After reading about five minutes the patient was forced to stop because of scotoma. During the past four months prior to the present admission he had noticed infrequent twitching of the muscles of the thighs and calves. When admitted to this hospital, he was unable to walk without assistance. Examination revealed an alert, cooperative patient with a short, thick neck, notably ataxic gait and moderate ataxia of the upper extremities. There were slight lateral nystagmus on his looking to either side and vertical nystagmus on upward vision. Both disks were elevated 1 D. Reflexes were physiologic, though generally exaggerated. There was no

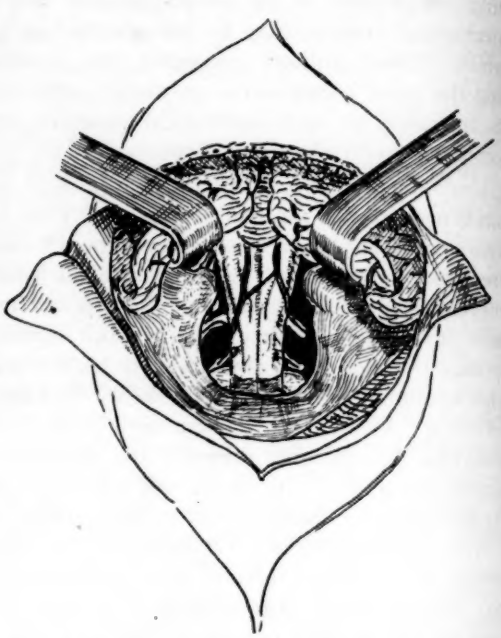

Fig. 3.—Removal of the arachnoid and elevation of the hemispheres to display two "ramps" of cervical spine elements projecting into the cerebellum.

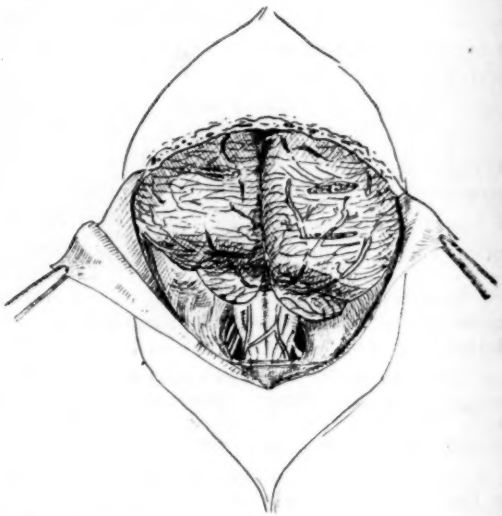

Fig. 4.—Final appearance. The cerebellum and the cervical portion of the cord are now free. The "ramps" of cervical spine elements have been removed. Patches of necrosis and atrophy of the cerebellum are evident.

evidence of motor weakness or sensory changes. Lumbar puncture revealed a partial block. The protein of the spinal fluid measured 156 mg. per hundred cubic centimeters. Roentgenograms of the skull at this time revealed, in addition to the postoperative defect in the occipital bone, persistence of cervical elements above Chamberlain's line.

Second Operation.—A second exploration was made on March 21, 1944. His old wound was well healed. The cistern was extremely large, extending upward and posterior to the cerebellum. The latter was small and atrophic, with patches of softening. A dense arachnoid was adherent over both cerebellar hemispheres, and the tonsils bulged from under this tight fibrous binder (fig. 2). On elevation of the inferior portion of the cerebellum, two "ramps" of cervical spine elements were seen to project upward through the occipital bone and into the hemispheres (fig. 3). When the arachnoid was removed and the "ramps" of the cervical spine elements were rongeured away, the wound rapidly filled with spinal fluid (fig. 4). There was no apparent pressure on the spinal cord.

Postoperative Course.—The postoperative course was uneventful. The patient states that the headaches and dizziness have been partially relieved. There is less ataxia in the upper extremities, but the patient complained at the time of his discharge from the hospital, on March 3, that control of his legs was not improved.

COMMENT

The case of basilar impression here presented is unique in several respects. The onset of symptoms at the age of 53 is unusually late. Most cases reported in the literature were discovered before the fourth decade of life. The popular explanation of this condition is that of a developmental anomaly. Consequent pressure of bony elements on the adjacent neural structures and mild chronic interference with the normal dynamics, said to be tolerated by the theoretically more robust nerve tissue of youth, become deleterious to older tissue. One is inclined to believe, however, that the aging process in a man past 50 displays more changes in the osteoarticular frame than in the ever soft and friable nerve tissues. Whatever may be the cause, a wide removal not only of the bony impingement but of a potentially constricting dura and arachnoid is essential if the nerve structures in cases of this condition are to be adequately decompressed.

It is difficult to determine at this time the extent of recovery to be expected in this patient. Grossly visible atrophy of the cerebellum was observed. However, in view of the widespread decompression and the satisfactory evidence that the nerve structures are now free, any subsequent progression of symptoms will suggest that the cerebellar degeneration is in itself progressive.

CONCLUSIONS

In a case of verified deformity of the occipito-atlas structure presented here, the associated neurologic signs were limited to those of a cerebellar lesion. Basilar impression, though rare, should always be considered in the differential diagnosis of a cerebellar tumor, as well as of such conditions as multiple sclerosis, syringomyelia, hydrocephalus and amyotrophic lateral sclerosis.

The importance of roentgenologic studies in cases of this anomaly cannot be overemphasized.

In the surgical decompression of basilar impression removal of all sources of compression, including any potentially constricting bone, dura or arachnoid, is advocated.

Presbyterian Hospital.

Abstracts from Current Literature

EDITED BY DR. BERNARD J. ALPERS

Physiology and Biochemistry

THE NATURE AND ORIGIN OF ALTITUDE PAINS. D. E. ROSENBLUM, Am. Rev. Soviet Med. **1**:303 (April) 1944.

Rosenblum subjected 13 men to thirty-five ascents, at an altitude of 21,000 meters, where they remained for from two to two and a half hours, after preliminary nitrogen desaturation. Ten subjects did not experience altitude pain, but they had had pain in almost all control ascents at lower altitudes.

A series of experiments was conducted on goats, dogs and rabbits, which were placed in an altitude chamber with a ceiling of 20,000 meters. Autopsy of the animals which died as a result of rapid decompression revealed evidence of acute caisson disease. There was no evidence of pseudocaisson disease, which follows rupture of the pulmonary parenchyma, with subsequent secondary air embolism.

Rosenblum concludes that the cause of altitude pain is caisson disease. Also, the data presented indicate that the coefficient of permissible nitrogen supersaturation of animal tissues is higher than the limit (2.25) established by Haldane for decompression from increased pressure.

GUTTMAN, Philadelphia.

HISTOMECHANICAL ANALYSIS OF NERVE REUNION IN THE RAT AFTER TUBULAR SPLICING. PAUL WEISS and A. CECIL TAYLOR, Arch. Surg. **47**:419 (Nov.) 1943.

Sectioned peripheral nerves in 39 rats were spliced by means of an arterial sleeve, and the subsequent reparative changes were studied histologically.

After reunion of the severed nerve ends by an arterial sleeve, a blood clot cements the approximated nerve ends. The red blood cells of the clot break up, leaving a fibrin net. The clot remains firmly attached to the nerve ends, but undergoes peripheral liquefaction and thus detaches itself from the arterial wall. This results in longitudinal tensions transmitted from nerve to clot to nerve and consequent longitudinal orientation of the fibrin fibers. A bridge of longitudinal strands of fibrin is formed between the nerve ends, whereas the remainder of the clot is dissolved by proteolysis. Macrophages clear the spaces between the strands of fibrin. Sheath cells move out from both nerve stumps, glide along the fibrin rails and deposit endoneural collagen on their surfaces. The axons regenerate in apposition to the sheath cells, following a straight, parallel course, without branching. This process of repair takes only a week; thereafter, the restitution of the nerve is completed by maturation and by increase in number of the newly formed nerve fibers.

In animal experiments, the method of nerve splicing with an arterial sleeve furnishes ideal conditions for nerve regeneration without the formation of neuromas and excessive scar tissue. Even though the method may not be directly suitable for human use, the histologic observations have revealed the clinically important fact that success or failure of nerve regeneration is determined within several days, or at most a few weeks, after the union of the nerve ends.

LIST, Ann Arbor, Mich.

NERVOUS REGULATION OF CLOTTING MECHANISM. GÉZA DE TÁKATS, Arch. Surg. **48**:105 (Feb.) 1944.

De Tákats studied the clotting mechanism by plotting in curve form the capillary coagulation time following administration of heparin. The normal heparin curve is biphasic. The clotting mechanism appears to be under the influence of the vegetative nervous system. Adrenergic stimuli, such as epinephrine, fear and apprehension, and hemorrhage increase the coagulation time and hence the tendency to thrombosis. Cholinergic stimuli, on the other hand, decrease the coagulation time, and therefore drugs such as prostigmine may be useful in preventing postoperative thrombosis.

LIST, Ann Arbor, Mich.

DIFFERENTIAL DIAGNOSIS OF PHYSIOLOGICAL AND PATHOLOGICAL SLEEP. LEO HESS, J. Nerv. & Ment. Dis. **98**:474 (Nov.) 1943.

In normal sleep the eyes are closed, the conjunctivas congested and the pupils small and sluggish in their reaction to light. The eyeballs are turned upward and inward; when light is thrown on them, they display lateral incoordinated movements. The face is reddened, especially the cheeks. Respiration is slow, usually regular, but occasionally periodic, and frequently of the costal type. Other features include slowing of the pulse, lowering of the blood pressure and hypotonicity of the musculature except for the orbicularis palpebrae. Waking is gradual, and there is no amnesia for the sleep. The need for sleep can be voluntarily resisted.

Miosis of the pupil occurs in normal sleep because of elimination of cortical inhibition of the Edinger-Westphal nucleus. It is also present in morphine-induced sleep.

Pathologic sleep and sleeplike conditions are associated with narcolepsy, hypnosis, alcoholism, morphinism, epilepsy, hyperpyrexia, diabetic and uremic coma, cerebral lesions and shock. These forms of sleep are not subject to the will; awakening is sometimes abrupt and often accompanied by lassitude and confusion rather than by a feeling of being refreshed. There may be amnesia for the sleep, as in cases of epilepsy. The face is pale, and breathing is irregular. Instead of muscular flaccidity, there may be motor signs, contractions and even convulsions. With deep unconsciousness the pupils are not miotic, and Bell's phenomenon is not present.

CHODOFF, Langley Field, Va.

ACTION OF METRAZOL ON THE MOTOR AND SENSORY NUCLEI OF THE BRAIN STEM. CARLOS GUTIERREZ-NORIEGA, J. Neuropath. & Exper. Neurol. **2**:132 (April) 1943.

After injection of minute doses of metrazol into or near the nuclei of the facial nerve of etherized cats which had been decerebrated through the diencephalon, myograms were obtained which recorded tonic and clonic contractions of the homolateral facial muscles. The relation of the magnitude of reaction to the proximity of the site of injection to the nucleus of the facial nerve supports the view that metrazol acts directly on the motor neurons.

The local action of metrazol on sensory neurons was studied by application of the drug to the nucleus of the spinal tract of the trigeminal nerve and recording the reflexly induced contractions of the ipsilateral temporal muscle. In some of the cats, tactile stimulation of the cornea resulted in clonic contractions of the ipsilateral superior labial and temporal muscles.

Injection of metrazol into the red nucleus or the mesencephalic tegmentum produced intermittent clonic contractions and running movements of the limbs.

These observations suggest that the convulsions which follow intravenous administration of minimal doses of metrazol are due to summation of a great number of local stimuli, those from sensory and supranuclear mechanisms augmenting and facilitating the direct action of the drug on motor neurons.

CAMPBELL, New York.

ACUTE PHYSIOLOGICAL EFFECTS OF GUNSHOT AND OTHER PENETRATING WOUNDS OF THE BRAIN. J. E. WEBSTER and E. S. GURDJIAN, J. Neurophysiol. **6**: 255 (July) 1943.

Webster and Gurdjian studied the effects of acute penetrating wounds of the brain in dogs. Gunshot injuries produced a sudden, intense increase of intracranial pressure, of short duration. The physiologic effects of the injury were classified as follows: (1) profound, with loss of respiratory and palpebral reflexes, hypertension and death; (2) moderately severe, with temporary interruption of respiratory and palpebral reflexes, rise in blood pressure and survival of some animals, and (3) minimal, with no significant change in the vital functions and with survival of all animals. These physiologic effects, the authors concluded, were the effects of the sudden increase in intracranial pressure.

FORSTER, Philadelphia.

EXPERIMENTAL HEAD INJURY PRODUCED BY BLASTING CAPS. S. R. GOVONS, Surgery **15**:606 (April) 1944.

Govons suggests the use of a blasting cap (du Pont no. 6 electric blasting cap) as a means of producing a severe type of standardized experimental head trauma. The method has the following advantages: The skull is not fractured; the animal is severely disabled with one blow; the cap can be applied to the freely movable head; the injury is confined to the head alone, and an easily measured and reproducible force is utilized. By use of this method, Govons was able to produce head injury in 9 animals which died immediately and in 4 animals which died within one-half to one and a half hours after the blast. Autopsy revealed changes varying from insignificant degrees of subarachnoid hemorrhage (5 animals) to severe intracerebral hemorrhage (2 animals). The majority of animals had petechial hemorrhages, usually in the brain stem and occasionally in the cerebral hemispheres.

The 17 animals which survived had a syndrome consisting of inability to stand, ataxia and salivation, which lasted from one to seven days. The final phase, consisting of difficulty in jumping, disturbance of gait and tendency to walk in a circle, lasted as long as eight months in 2 of the animals. The difficulty in jumping was attributed to the absence of the labyrinthine righting reflexes, as indicated by the absence of postrotatory nystagmus and of reflex extension of the limbs of the blindfolded injured animal when it was dropped through space. The gross necropsy observations were inconsistent, the majority of the animals showing only small petechiae in the brain stem and superficial contusion of the cortex at the site of impact. In 4 animals no gross evidence of contusion or hemorrhage was seen.

The author concludes that the disturbance uniformly produced was due to damage to the labyrinths and suggests that the labyrinths may play a large part in the mechanism of cerebral concussion, as a result of labyrinthine stimulation at the time of concussion, with the setting off of a widespread reflex inhibition of the central nervous system.

SHENKIN, Philadelphia.

MEASUREMENT OF VIBRATION SENSE. AARON ROTH, War Med. **4**:280 (Sept.) 1943.

Roth describes a new precision instrument, the neurometer, which is a tuning fork standardized to a frequency of 128 cycles, an intensity of 70 decibels at the maximum and a perception time of thirty-five seconds at the fingers. It has a cross bar in the stem.

With this instrument a special regional threshold for vibration sense for each part of the body can be determined, as a result of which it is possible to detect small fields of impairment. Since the author found that vibratory sensation in the fingers is lowered by cold, general fatigue and strenuous exercise of the fingers, he believes that this simple instrument may be of value in estimating the fatigue level for flight personnel.

PEARSON, Philadelphia.

Neuropathology

GENESIS OF ENCEPHALOPATHY DUE TO ARSPHENAMINE. I. MARK SCHEINKER, Arch. Path. **37**:91 (Feb.) 1944.

Scheinker reports 5 cases of encephalopathy due to arsphenamine, in all of which the essential alterations consisted of vascular changes typical of central vasoparalysis, associated with focal hemorrhagic and nonhemorrhagic lesions in the nerve parenchyma. The vascular lesions are interpreted by the author as the result of an acute physical or chemical irritant applied to the vessel walls which is propagated along the affected vessel. A contraction of the vessel wall of short duration is followed by paralytic dilation. This is usually associated with blood stasis, accumulation of carbon dioxide and, hence, further vascular dilation. The retarded circulation interferes with an adequate supply of oxygen to the smaller veins and capillaries, and if the alterations are severe, degeneration of the vessel wall may occur. The vessel wall then becomes permeable for serous fluid and red blood cells. The resulting extravasation into the distended perivascular spaces accounts for the punctate hemorrhages.

The author suggests that the term "encephalopathy due to arsphenamine" be substituted for "hemorrhagic encephalitis" or "brain purpura."

WINKELMAN, Philadelphia.

ARACHNOIDAL FIBROBLASTOMA (MENINGIOMA) WITH METASTASES TO THE LIVER. JOHN B. HAMBLET, Arch. Path. **37**:216 (March) 1944.

Primary meningeal tumors with extracranial metastases have been reported infrequently. Two previous cases of arachnoidal fibroblastoma with metastasis to distant regions have been reported.

The author reports the case of a 41 year old Chinese with clinical evidences of a tumor of the frontal lobe. Exploration revealed a tumor weighing 225 Gm., which was removed in eight pieces. After operation the patient's blood pressure fell rapidly and respiration ceased.

At autopsy the site of operation in the brain showed only remnants of ragged tumor tissue firmly attached to the cranial dura of the anterior fossa. The liver contained eight scattered masses, measuring from 0.3 to 3 cm. in diameter. The structural features of the tumors of the brain and of the liver were identical and were considered to be typical of arachnoidal fibroblastoma.

WINKELMAN, Philadelphia.

DIFFUSE MENINGEAL SARCOMA. ANTONIO ROTTINO and ROBERT POPPITI, J. Neuropath. & Exper. Neurol. **2**:190 (April) 1943.

A girl aged 10 years, with one year's history of symptoms and with neurologic evidence of an expanding lesion of the right parietal lobe, suddenly became comatose and died. Gross examination of the brain revealed a thick, plaquelike, nonencapsulated tumor occupying the leptomeninges and the cortex of the dorsolateral surface of the right parietal lobe, and adherent to the overlying dura.

Microscopic examination of the tumor revealed that the type and arrangement of the cells were characteristic of meningioma, but in some areas multinucleated giant cells and abundant mitotic figures justified the diagnosis of meningeal sarcoma. Most notable was the fact that numerous columns of cells from the under surface of the neoplasm penetrated the cortex along the course of the blood vessels. The tumor was thickest centrally, where the brain was massively infiltrated. At its periphery the meningeal growth gradually thinned, to become confined to the pia-arachnoid. Elsewhere in the meninges there were focal areas of solid membrane.

The author believes that this case of a malignant meningeal tumor gives support to the belief expressed by Globus that "that in certain conditions, the meninges covering the brain may retain their premature arrangement after birth," and to Weinberger's theory that "the presence of such sheets of undifferentiated tissue would *a priori* provide a source for future tumor formation."

CAMPBELL, New York.

SIDEROSIS OF THE GLOBUS PALLIDUS IN A MONKEY. P. GLEES, J. Neurol. & Psychiat. **6**:92 (July-Oct.) 1943.

Glees reports bilateral siderosis in the globus pallidus of a 4 year old male monkey which had been treated with injections of estrone monobenzoate. The deposits of iron had no relation to the blood vessels and were associated with considerable damage to the parenchyma of the pallidum. In spite of this, no clinical signs of pallidal disease were apparent. The condition is interpreted as a metabolic dysfunction of the globus pallidus, and the question is raised whether this was related to the treatment with estrone.

MALAMUD, Ann Arbor, Mich.

Diseases of the Brain

CHRONIC SEASICKNESS. ROBERT S. SCHWAB, Ann. Int. Med. **19**:28 (July) 1943.

Schwab classifies seasick patients under three types:

Type 1.—Persons of this type suffer from a variety of forms of motion sickness and become sick not only at sea but on trains, buses, cars, elevators, swings and roller coasters. When such persons become seasick, they are extremely sick and stay sick.

Type 1A.—Persons of this type have a history of motion sickness, but in quiet water or on a big ship get along reasonably well.

Type 2.—Persons of this type have no history of motion sickness. They suffer from seasickness particularly during rough weather.

Of the patients of the first type, 74 per cent showed neurotic trends of one sort or another, and many manifested a definite psychoneurosis. Persons of this type, with neurotic trends should, in the experience of the author, be discharged from the Navy. The rest of the patients of type 1, if valuable to the service, should be recommended for shore duty. The patients of type 2 may be helped by sedatives, atropine and related drugs or by transfer to a larger ship.

Of the patients suffering from chronic seasickness, 50 per cent showed abnormalities of the gastrointestinal tract. The gastrointestinal abnormalities were (1) pronounced irritability of the pylorus and duodenum, with resulting pyloric spasm; (2) marked increase in gastric secretion; (3) some increase in the gastric rugae, and (4) loss of peristalsis. Fifteen patients examined with the gastroscope showed evidence of mild, chronic superficial gastritis.

PRICE, Philadelphia.

TRIGEMINAL NEURALGIA DUE TO RADICULAR LESIONS. W. B. HAMBY, Arch. Surg. **46**:555 (April) 1943.

Lesions in the vicinity of the intracranial portion of the fifth nerve may cause trigeminal pain, which may assume the character of true tic douloureux. Tumor or aneurysm in the middle fossa, compressing the gasserian ganglion and its peripheral branches, occasionally causes a ticlike pain, but as a rule there is also a background of steady, burning pain and numbness. Neoplasms of the cerebellopontile angle encroach on the sensory root of the trigeminal nerve and may produce a pain which is indistinguishable from that of idiopathic trigeminal neuralgia. Since in cases of such tumors trigeminal pain may be the first or the most conspicuous symptom, evidence of involvement of other cranial nerves must be carefully sought for.

The author reports 4 cases of cerebellopontile neoplasm (2 of epidermoid, 1 of meningioma and 1 of acoustic neuroma), in which at first only the diagnosis of trigeminal neuralgia was made and the signs of a lesion in the cerebellopontile angle were minimal and late in appearance. It is worthy of mention that peripheral injection of alcohol or customary root section by the temporal approach may relieve pain caused by a lesion of the trigeminal root in the posterior fossa. Every patient with trigeminal neuralgia should be carefully examined for the possible presence of a lesion in the posterior fossa, especially if the patient is young. Under such circumstances, it is advisable to perform trigeminal rhizotomy by Dandy's posterior approach, lest gross lesions of the cerebellopontile angle be overlooked. For the same reason, the cerebellopontile angle should be explored when a trigeminal tractotomy is to be performed in the medulla. Although trigeminal rhizotomy by the cerebellar route is safe in skilled hands, the subtemporal approach is preferable for the treatment of trigeminal neuralgia in the elderly patient, who is usually a poor operative risk.

LIST, Ann Arbor, Mich.

PSEUDARTHROSIS AND NEUROFIBROMATOSIS. W. T. GREEN and N. RUDO, Arch. Surg. **46**:639 (May) 1943.

Green and Rudo report the case of a girl aged 7 years who had neurofibromatosis. She presented cutaneous manifestations of the disease (café au lait spots), a plexiform neuroma on the right radial nerve and neuro-

fibromatous lesions of the right tibia, with resultant bowing of the leg and pseudarthrosis of the fibula. The child was under observation for two and a half years, during which time four operations were performed. The neurofibromatous lesions of the bone recurred repeatedly after partial excision, and two pathologic fractures of the tibia developed, with formation of pseudarthrosis. Finally, the areas of pseudarthrosis healed solidly, after plating had been done with bone grafts.

Histologic examination showed that the neurofibroma of the bone originated from intraosseous, rather than from periosteal, nerves. The presence of recurrent neoplasm after incomplete excision was undoubtedly responsible for the spontaneous fractures and for the retardation or prevention of a bony union. In cases of congenital pseudarthrosis the possibility of an intraosseous neurofibroma should be kept in mind and other evidence of neurofibromatosis (Recklinghausen's disease) should be sought for.

LIST, Ann Arbor, Mich.

SUBDURAL EMPYEMA. CHARLES S. KUBIK and RAYMOND D. ADAMS, Brain **66**:18 (March) 1943.

Subdural empyema denotes a collection of pus in the subdural space. Kubik and Adams report 14 cases. In 12 cases the condition followed infection of the paranasal sinuses, in 1 case it was secondary to infection of the ear, and in 1 case it was metastatic from a chronic suppurative lesion in the lung. In 12 cases the outcome was fatal, and in 2 there was recovery; in both the latter the cerebral abscess resulted from infection of the sinuses. Thirteen of the 14 patients were males, and 8 of the 14 patients were between the ages of 12 and 30 years, the ages of the others varying up to 55 years. Subdural empyema usually follows an acute episode of a chronic infection, though in 1 of the reported cases it was apparently secondary to primary acute sinusitis. Chronic frontal sinusitis with frontal osteomyelitis and epidural infection is particularly favorable for its development.

The onset is marked by local pain which is referred to the region of the affected ear or sinus, followed by generalized, severe, persistent headache with nausea and vomiting. This is accompanied by a pronounced rise in temperature. The increased temperature is associated with increased intracranial pressure and rigidity of the neck. Sooner or later the patient becomes confused or somnolent, the condition progressing rapidly to stupor or coma. At about the same time focal neurologic signs begin to appear, the most important being jacksonian seizures (seen in 50 per cent of cases), hemiparesis, aphasia and paralysis of contralateral conjugate deviation of the eyes. In the fatal cases death occurred two to five days after the appearance of focal signs. Roentgenograms usually give evidence of sinusitis and, in some instances, of frontal osteomyelitis. The cerebrospinal fluid is under moderately increased pressure, and there is pleocytosis of moderate degree, with predominance of polymorphonuclear cells. Bacteria were never demonstrated in smears or by culture.

With the onset of focal signs, sinus thrombosis and cerebral abscess must be differentiated. In the former condition there is seldom a stiff neck, and the signs are bilateral. Cerebral abscess has a milder, more insidious, onset, and the course is usually more protracted. The temperature is seldom high, and the cell count of the spinal fluid is normal, or only slightly increased; lymphocytes predominate. In 4 of the 7 cases complicating sinusitis the subdural abscess occurred by direct extension through the dura following osteomyelitis of the frontal bone; in 2 cases it followed necrosis or perforation of the bony wall of the frontal sinus or the ethmoid cells and dura, and in 1 case it followed an operative fracture. In the remaining cases it followed thrombophlebitis of a venous sinus. The subdural exudate usually covered a large part of one cerebral hemisphere. The exudate was thickest over the lower lateral portion of the frontal lobe, extending to the orbital and medial surfaces, over the temporal lobe and as far back as the parietal lobe. It was never walled off. A subarachnoid exudate was limited to the area covered by the subdural collection. There was rarely generalized leptomeningitis.

Microscopic studies revealed thrombosis or thrombophlebitis of the adjacent subarachnoid veins and severe ischemic necrosis of the subjacent cortex.

Treatment consists of surgical drainage as soon as the diagnosis is made. Drainage is best accomplished through an enlarged burr hole in the lateral frontal region, since this is the usual site of the greatest accumulation of exudate. This approach provides an attack through a clean field and results in no harm if exploration gives negative results. The dura over a subdural abscess is white and easily recognized. The use of sulfonamide compounds is recommended.

SHENKIN, Philadelphia.

ON THE SO-CALLED "LARYNGEAL EPILEPSY." C. W. M. WHITTY, Brain **66**:43 (March) 1943.

Whitty reports 4 cases of laryngeal epilepsy, a condition occurring in middle-aged men of plethoric type in association with laryngitis or bronchitis. A previous personal or family history of epilepsy is uncommon. The presenting symptom is an attack of dizziness or loss of consciousness, preceded by a tickling sensation in the larynx and cough, which may be slight or severe. Epileptiform movements may occur during the attack, but a more constant feature is slight mental confusion following the attacks. In about one fourth of the cases apparent recovery or remission follows local treatment of the upper respiratory tract.

The author found that forced expiration with a closed glottis failed to produce asystole or convulsions in any of his cases. That cerebral venous congestion and anoxemia due to cough were etiologic factors was regarded as unlikely, since venous constriction failed to produce an attack in any case in which forced coughing produced an aura. Furthermore, it was found that in a case in which venous obstruction produced an aura, attacks were independent of coughing. The author concludes, therefore, that in the majority of cases the condition is primarily neurogenic and represents a form of genuine epilepsy.

The neurogenic factor which appeared to be at play in the cases reported was true reflex laryngeal epilepsy, epilepsy with a laryngeal aura or venous congestion caused by violent coughing in a case in which the cerebral cortex was predisposed to epilepsy, whether by vascular degeneration or by other factors.

SHENKIN, Philadelphia.

HEMIPLEGIA COMPLICATING WHOOPING COUGH. L. J. GRANT and C. E. WILLIAMS, Brit. M. J. **2**:9 (July 3) 1943.

Although hemiplegia is a rare complication of whooping cough, it has been estimated that this phenomenon occurs in about 50 per cent of cases of pertussis associated with neurologic conditions, exclusive of convulsions. The neurologic complications are ascribed by many authors to "encephalitis," but Grant and Williams

believe that this is not universally true, as the diagnosis is often made from unreliable pathologic data. Other investigators have explained the complication on the basis of focal intracranial hemorrhage. In the authors' estimation, the most plausible theory is that the hemiplegia is sometimes caused by air embolism. The case which they report seems to support this view. A 5 year old boy had whooping cough associated with subcutaneous emphysema, which was finally complicated by permanent hemiplegia. The subcutaneous emphysema was thought to originate in acute interstitial emphysema, which diffused to the neck by way of the posterior mediastinum. The authors believe that air may have been liberated into the circulation at any time during the period of formation of the parenchymatous emphysema, to produce the paralysis.

ECHOLS, New Orleans.

MIXED ADENOMA OF THE HYPOPHYSIS. CARLOS GAMA, Arq. Inst. Penido Burnier **6**:1, 1940.

Gama recounts the history of surgery of the hypophysis up to the present and gives his own statistics on the results of operations in the region of that gland. Of 114 craniotomies performed for tumor, only 8 were for hypophysial tumor. The author reports in detail the history of a woman aged 46 on whom he operated for hypophysial tumor by the temporofrontal, intradural route. The pathologic diagnosis was mixed adenoma. A photomicrograph of the tumor is given, but neither the text nor the illustration makes it evident on what this diagnosis was based. The patient survived the operation, and the visual defects receded. There were no signs of acromegaly. The author advises against section of an optic nerve for a better view of the tumor, since the present case shows that an amaurotic nerve may recover some function.

BAILEY, Chicago.

Meninges and Blood Vessels

THE RELATION OF THE CARRIER TO EPIDEMIC MENINGITIS. J. HOWARD MUELLER, Ann. Int. Med. **18**:974 (June) 1943.

Mueller discusses the difficulty in tracing direct contacts between patients with epidemic meningitis. He assumes this is because only an occasional person who harbors the organism is actually infected. The same virulent strain may, therefore, be passed from one healthy carrier to another through many hosts before it reaches one in which the conditions for invasion are suitable. By the time that this occurs all trace of the intervening chain of passage has been lost.

The author found that the total carrier rate was remarkably constant, showing no evidence of seasonal variation. In another community of persons living in relatively close contact during the course of a rather sharp outbreak of infection with type 1 meningococcus, between 60 and 70 per cent of several hundred for whom cultures were made were carriers of type 1 meningococci.

Of approximately 400 persons, one-half were given sulfadiazine on three successive days: 3 Gm. the first day and 2 Gm. on each of the following days. The other half served as a control. Three days after the drug was given, cultures were made of secretions from the upper respiratory tract of all the persons so treated. In the control group the carriers of type 1 meningococci increased from 68 to more than 70 per cent. Of the 200 persons who had received sulfadiazine, not one carried a meningococcus of any sort. As actual quarantine is probably out of the question, it appears that in an emergency it may be possible to eradicate carriers from a given group of persons, at least temporarily, by the use of sulfadiazine.

PRICE, Philadelphia.

FULMINATING MENINGOCOCCIC INFECTION WITH BILATERAL MASSIVE ADRENAL HEMORRHAGE (THE WATERHOUSE-FRIDERICHSEN SYNDROME). HARRISON S. MARTLAND, Arch. Path. **37**:147 (Feb.) 1944.

The series of 19 cases of the Waterhouse-Friderichsen syndrome analyzed in this report is perhaps the largest observed by one person, and the author's conclusions are based entirely on his personal experience. 1. The Waterhouse-Friderichsen syndrome (cyanosis, purpura, petechias and bilateral massive hemorrhages in the adrenal glands without gross meningitis) is always that of fulminating, fatal meningococcemia, in which death occurs before there is notable invasion of the leptomeninges. 2. This condition is not as rare as the literature suggests and is not limited to infants or children, but is apt to be encountered in adults whenever meningococcic infections are prevalent. 3. The clinical and anatomic diagnosis is comparatively simple after attention has been called to the condition. 4. The hemorrhages in the adrenal glands apparently are due to several factors: the toxins liberated by the enormous number of meningococci, the selective affinity of this toxin for capillary endothelium and the presence of a susceptible rich, delicate capillary network in the adrenal cortex. However, death is due to the overwhelming meningococcemia rather than to acute insufficiency of the adrenal cortex. 5. On account of the frequency of sudden and unexpected death, the sporadically occurring Waterhouse-Friderichsen syndrome in civilian life must sometimes be differentiated from food or chemical poisoning, trauma, negligence and even homicide.

WINKELMAN, Philadeltphia.

ACUTE LYMPHOCYTIC MENINGITIS IN "EPIDEMIC CATARRHAL JAUNDICE." J. WARING, Brit. M. J. **2**:228 (Aug. 21) 1943.

Waring believes that the terms "epidemic catarrhal jaundice" and "infective hepatitis" are misnomers because these conditions are in reality due to a general virus infection, such as influenza, in which the primary site of invasion is frequently the gastrointestinal tract. He suggests that the term "influenza" be used in a generic sense to include these conditions rather than that its use be restricted to one virus infection. He points out that "catarrhal jaundice" and infective hepatitis give a false impression of the disease they represent because they exclude the diarrheal type. Thus, in an epidemic of virus infection primarily affecting the gastrointestinal tract there was approximately 1 case of jaundice to every 6 cases of diarrhea. The epidemic disease was observed to have three distinct forms: the diarrheal, the vomiting and the jaundiced. Waring reports in detail a case of acute lymphocytic meningitis occurring in the course of influenzal jaundice.

ECHOLS, New Orleans.

A CASE OF ACUTE OTOGENOUS LABYRINTHITIS AND MENINGITIS. MAXWELL ELLIS, Lancet **1**:468 (April 8) 1944.

Ellis reports the case of an airman aged 18 who was successfully treated with sulfapyridine for nonhemolytic streptococcic labyrinthitis and meningitis. Recovery was eventually complete except for absolute deafness and loss of labyrinthine responses on the affected side. The author emphasizes the need for fearless use of the drug to achieve entire freedom from infection. If

the bone marrow is coincidentally depressed, a large blood transfusion, repeated as often as necessary, is both a rational and a safe antidote. It is practical to concentrate on elimination of the infection and to worry about changes in the blood if and when they occur.

McCarter, Philadelphia.

Vegetative and Endocrine Systems

Gargoylism. Louis A. Lurie and Sol Levy, Am. J. M. Sc. **207**:184 (Feb.) 1944.

Gargoylism is a syndrome complex which is characterized by chondrodystrophic skeletal changes and the deposition of a lipid-like substance in many of the body tissues. Lurie and Levy report 2 cases in brothers, the first of whom was 7 years and 8 months and the second 6 years and 9 months of age. Both patients showed the characteristic skeletal deformities, pronounced enlargement of the spleen and liver, umbilical hernia and feeblemindedness.

From the pathoanatomic standpoint, gargoylism has been placed among the lipoidoses, abnormal storage of lipid-like substance being observed in various tissues, principally the reticuloendothelial cells, the brain, the cornea, the pituitary gland, the liver, the spleen and the lymph nodes.

Michaels,
Medical Corps, Army of the United States.

Surgical Removal of Adrenal Adenoma with Relief of Cushing Syndrome. Seymour F. Wilhelm and Sidney Gross, Am. J. M. Sc. **207**: 196 (Feb.) 1944.

Wilhelm and Gross report the case of a man aged 46 whose general appearance suggested a Cushing syndrome (pituitary basophilism), with notable adiposity of the face, neck and abdomen. The face and neck were dusky red, and there were purplish striae on the abdomen. A simple roentgenogram and laminogram revealed a large tumor in the region of the right adrenal. Operation revealed an encapsulated tumor, about the size of an orange, directly above the kidney. The tumor was lobulated and was composed of coarse strands several cells in thickness, separated by a delicate connective tissue membrane. Eventually the patient improved, after a second operation five weeks after the first operation for removal of a spherical fatty tumor, 7.5 cm. in diameter, which lay between the peritoneum and the deep fascia of the rectus muscle. Postoperative treatment was directed toward prevention of adrenal cortex insufficiency. Frequent blood pressure readings served as an important guide to the administration of adrenal cortex extract, desoxycorticosterone and infusions of isotonic solution of sodium chloride.

About three months after removal of the first tumor the patient said he was well but that he tired after heavy exertion. The systolic pressure was 128 and the diastolic 85. He was completely relieved of his symptoms after extirpation of the tumor and has remained well for over sixteen months since the operation.

Michaels, Medical Corps,
Army of the United States.

Environmental Modification of Certain Endocrine Phenomena. Edward W. Dempsey and Harold F. Seares, Endocrinology **32**:119 (Feb.) 1943.

Study of the vaginal cycles of a group of female rats after transection of the pituitary stalk showed that unaltered, normal cycles were demonstrable in approximately half the animals. Pseudopregnancy, as indicated by prolonged diestrus, production of deciduoma and short periods of vaginal cornification after injection of the estrogen, were produced in rats with section of the pituitary stalk. Approximately one fifth of the animals displayed prolonged diestrous intervals. The prolonged cycles, however, could not be characterized as pseudopregnant cycles, since tests for luteal function gave negative results. Maintenance of female rats in variously altered environments has shown that constant light causes constant follicular development without ovulation. Ovulation and pseudopregnancy were induced, however, by sterile mating. High environmental temperatures caused a depression in the height of the thyroid cells and enlarged adrenals. Simultaneous exposure to cold and light produced constant estrus, as well as stimulation of the adrenals and thyroid. All these reactions, except for stimulation of the thyroid by cold, were produced in animals after section of the pituitary stalk. The results show that environmental stimuli may be transmitted to the anterior lobe of the pituitary by routes other than the pituitary stalk. Furthermore, they demonstrate that various endocrine components react in unitary fashion when the endocrine balance is altered.

Palmer, Philadelphia.

Influence of Sex and Sex Hormones on the Breaking Strength of Bones of Mice. W. U. Gardner, Endocrinology **32**:149 (Feb.) 1943.

The breaking strength of the femurs of 241 mice which had received estrogens, such as estradiol benzoate, plus testosterone propionate and the breaking strength of the femurs of 162 untreated mice were determined, under uniform conditions. The femurs of the untreated mice decreased in strength as age increased. The strength of the femurs of the females was greater than that of the males. In general, the most radiopaque femurs showed the greatest resistance to fracture. The breaking strength of the femurs was much greater in the estrogen-treated mice than that of the femurs of the controls of the same age groups. Estrogen-treated male mice had stronger femurs than females similarly treated. The bones of the old estrogen-treated mice were relatively stronger than the bones of the younger estrogen-treated mice as compared with controls of the corresponding age groups. The simultaneous injection of testosterone propionate and estrogen prevented the increased strength observed in mice given estrogen alone. The bones of female mice given testosterone propionate were much weaker than the bones of untreated controls of the same age groups. The presence of the ovaries or testes or the injection of estrogen or androgen influenced the strength of the bones of mice.

Palmer, Philadelphia.

Stimulation of Mammary Glands in Hypophysectomized Rats by Estrogen and Testosterone. Samuel L. Leonard, Endocrinology **32**:229 (March) 1943.

Leonard noted stimulation of the end buds of the mammary glands in immature rats between the ages of 22 and 30 days, weighing not over 70 Gm., after hypophysectomy and immediate injection of adequate doses of estradiol dipropionate. The injections were continued over a period of ten to twelve days. The degree of stimulation was small as compared with the results obtained with normal or with partially hypo-

physectomized rats; yet the significance of the results is increased since stimulation of end buds was not obtained when the rats were older and heavier at the time of hypophysectomy or when the injections were delayed after the operation, regardless of the age and weight. Injections of testosterone propionate into young hypophysectomized male rats resulted uniformly in thickening of the ducts of the mammary tree. This effect was due to hyperplasia and hypertrophy of the epithelial cells and an increase in the diameter of the lumen of the ducts. Secretory material was observed in many ducts of the stimulated glands and could be seen forming from the cytoplasm of the epithelial cells. This observation indicates a possible mechanism of the synergistic action of testosterone and the pituitary mammogen, in which the mammogen not only augments the effects of testosterone but permits lobular-alveolar growth to occur.

PALMER, Philadelphia.

INFLUENCE OF AGE ON THE EFFECT OF THYROIDECTOMY IN THE RHESUS MONKEY. W. F. FLEISHMANN, H. B. SHUMAKER JR. and W. STRAUS JR., Endocrinology **32**:238 (March) 1943.

The authors present additional data on the effects of thyroidectomy in rhesus monkeys to supplement knowledge of the effects of thyroid insufficiency in man and to clarify some of the conflicting reports from the older literature. In 6 adult rhesus monkeys total thyroidectomy had no effect on the appearance or the activity of the animals or on the level of cholesterol in the serum. The basal metabolic rate showed no change in 2 monkeys for which it was determined. The animals survived from forty-four to four hundred and eighty-seven days. The completeness of the thyroidectomies was demonstrated not only by anatomic study but by absence of response to a preparation containing thyrotropin and a definite response to thyroxin. Two additional monkeys were thyroidectomized at 8 months of age. Both animals showed a sharp rise in the cholesterol content of the serum after operation. Complete cessation of growth was observed in the larger, surviving animal. The authors suggest that age is of great importance in determining the effect of thyroidectomy in the rhesus monkey. Since the thyroid gland of the adult rhesus monkey shows histologic signs of activity, it is difficult to assume that it has lost its part in the regulation of metabolism. It is more probable that some complementary regulatory mechanism is normally present in the adult of this species and is capable of taking over the function of the gland after its removal.

PALMER, Philadelphia.

CONSTANCY OF ANDROGEN CONCENTRATION IN THE URINE. FRANKLIN HOLLANDER, BRUNO KRISS, EMANUEL KLEMPNER and ROBERT T. FRANK, Endocrinology **33**:216 (Oct.) 1943.

Contrary to observations previously reported by Gesting and Webster, data are presented which indicate good correlation between the daily androgen output and the daily urinary volume. The content of biologically active androgens and the volume of urine were determined for a series of consecutive three day specimens of urine collected for approximately one month from each of 9 normal human subjects. In 3 patients the urinary output was varied throughout the period of study by forced and restricted intake of fluids. The results show that the concentration of biologically active androgens in the urine is constant within the limits of reliability of the method of assay and is independent of the daily urinary output, even under forced and restricted intake of fluids. The variations in daily output of androgens paralleled the daily output of urine. The constancy of concentration is not in accord with the behavior of any other urinary constituent.

PALMER, Philadelphia.

INHIBITING EFFECT OF ADRENOCORTICOTROPIC HORMONE ON THE GROWTH OF MALE RATS. HERBERT M. EVANS, MIRIAM E. SIMPSON and CHOH HAO LI, Endocrinology **33**:237 (Oct.) 1943.

The authors present data to show the inhibition of growth by the adrenocorticotropic principle in normal and in gonadectomized male rats. They used an extract from the sheep pituitary containing the principle in partially purified form. The inhibition does not occur in adrenalectomized rats.

PALMER, Philadelphia.

HORMONE FACTORS IN MALE BEHAVIOR OF THE FEMALE RAT. RUDOLF KOSTER, Endocrinology **33**:337 (Dec.) 1943.

Koster discusses the sexual behavior of normal, castrated and androgen-treated female rats. Female rats, given injections of testosterone from birth, copulated on estrous females and were also attractive to normal males, who copulated and showed spasmic reactions frequently in spite of the abnormal female anatomy. One such treated female copulated on 2 normal males in spite of strong resistance. Females given injections of estradiol from birth showed strong masculine behavior. Masculinity persisted long after the injections were discontinued, and approximately 2,000 rat units was necessary for reversal. Normal males were strongly attracted to these females but were actively resisted by them. The authors conclude that in sex reversal the estrogen upset the established organization of behavior and stimulated the function of other sensorimotor routes which are normally suppressed. In this reorganization of behavior patterns the pituitary is probably not involved, and the estrogen probably acts directly on some part of the central nervous system. The female rat is genetically endowed with the nervous and muscular organization for both male and female behavior, and these two systems can function concurrently in the same animal.

PALMER, Philadelphia.

EFFECT OF THIOURACIL AND THIOUREA ON THE THYROID GLAND OF THE CHICK. J. P. MIXNER, E. P. REINEKE and C. W. TURNER, Endocrinology **34**:168 (March) 1944.

The authors found that thiouracil and thiourea produced enlargement of the thyroid of chicks. Maximum enlargement was obtained in twelve days, when a 0.1 per cent solution of thiouracil was given in the diet to 2 day old white Plymouth Rock chicks. The female chicks showed the greater response as measured by the weight of the thyroid gland after administration of thiouracil. Greater amounts of thyroxin were also required by the female chicks to depress the weight of the thyroid to any given level. It was found that different breeds of chicks exhibited different responses to thiouracil, but the response in the weight of the thyroid was fairly constant within a given breed. The thyroid of the lighter breeds gave a greater response than the thyroid of the heavier breeds.

PALMER, Philadelphia.

Society Transactions

PHILADELPHIA NEUROLOGICAL SOCIETY

GEORGE D. GAMMON, M.D., Presiding

Regular Meeting, Jan. 28, 1944

Sacrococcygeal (Pilonidal) Sinus in Direct Continuity with the Central Canal of the Spinal Cord: Report of a Case. DR. HENRY A. SHENKIN, DR. ANDREW D. HUNT JR., and DR. ROBERT C. HORN JR.

A sacrococcygeal (pilonidal) sinus in a 2 year old child was demonstrated at autopsy to be in direct continuity with a patent and dilated filum terminale, which, in turn, was continuous with the central canal of the spinal cord. The clinical picture of transverse myelitis was due to an abscess about the central canal of the lumbar portion of the cord. Death was due to widespread leptomeningitis, caused by a microaerophilic nonhemolytic streptococcus.

The case is offered as evidence that sacrococcygeal sinus originates as a malformation in the development of the neural canal. The importance of early excision of such a congenital lesion is indicated by the development in this case of an intramedullary abscess of the spinal cord and purulent meningitis as a result of contamination through the dermal sinus.

DISCUSSION

DR. ROBERT C. HORN: Sacrococcygeal, or pilonidal, sinus is common in the experience of the general surgeon, and therefore in that of the general surgical pathologist. Within the last year or so the lesion has taken an important place in the general surgical literature because of the large number of cases that have been reported, chiefly from various Army camps. This large incidence has been attributed to the fact that the population of the camps is made up of men of a limited age group, who are vigorous and active; furthermore, the mechanization of the modern army results in a type of trauma which predisposes to infection, the lesions of which come to the attention of the patient and the surgeon.

The articles in the literature have been primarily concerned not with the origin of the lesion but with the treatment, preferably such treatment as will allow the medical corps to get the soldiers back to duty in as brief a time as possible.

The great majority of pilonidal sinuses which are seen in a general pathologic laboratory do not differ from dermoid cysts situated elsewhere except for the frequent occurrence of secondary infection, with destruction of many of the specific characteristics.

Occasionally, one of these sinuses is attached to the sacrum by a bond which usually is simply fibrous tissue, and it has no other connection with the central nervous system. The case presented by Dr. Shenkin and his associates is the first in my experience in which a sinus of this kind was directly connected with the central nervous system.

DR. FRANCIS C. GRANT: I was not interested so much in the embryologic features of this case as I was perturbed because the urologist and the pediatrician had, in my opinion, missed the proper diagnosis. The child had had urinary retention for thirty hours and diplegia for twenty-four hours. An infectious process was present, presumably in the sacral area. Neurosurgeons who have had any experience with diplegia as a result of an infectious process about the cord know, unfortunately full well, that if complete paralysis is permitted to develop the patient rarely recovers much power.

I feared that the pediatrician and the urologist had permitted this child to reach the stage in which operation was useless. The surgical treatment was unsuccessful, but it appears from the autopsy evidence that even if the child had been treated earlier, immediately after the development of urinary retention, the outcome would not have been different.

An amazing series of cases of epidural lesions has occurred in the University Hospital during the last twelve months—lesions which I did not believe existed. There were 2 cases of epidural cyst with no connection with the dura—simple cysts lying loose in the epidural space—1 case of chronic subdural hemorrhage within the spinal dura and 2 cases of acute epidural hemorrhage, in both of which hypertension was present. One or 2 cases of epidural abscess also occurred.

Since I had felt that this patient should have been operated on earlier, it was a relief that Dr. Shenkin could prove direct connection between the abscess and the pilonidal cyst and, furthermore, that the infection had traveled up the central canal of the cord. Even if this patient had been seen sooner, the chances are that the situation was hopeless from the beginning. In cases in which there is a possible source of infection, with development of retention of urine and pain in the back, followed by motor and sensory disturbances, the chances are good that there is epidural infection of the spinal cord. Unquestionably, the sooner the operation, the better the prospect of recovery.

DR. GABRIEL SCHWARZ: A good deal of interest has been shown recently concerning the relation of maldevelopment of the spinal cord to maldevelopment of the overlying skin, muscle and bone. This group of anomalies has been termed myelodysplasia. Since Dr. Shenkin's patient had an abscess which involved mainly the central portion of the spinal cord, I wonder whether there was any evidence of dysplasia in the portions of the spinal cord affected by the abscess, or even in sections higher than the extent of the abscess. May there not have been syringomyelia with secondary infection of the region of malformation from the sinus below?

DR. HENRY A. SHENKIN: I believe that this condition was not syringomyelia for the reason that immediately adjacent to the abscess of the spinal cord the central canal was intact and it could be seen to be filled with inflammatory debris. I see no reason to think that this condition was anything else than an infection ascending from an already infected, dilated filum terminale.

Infected Subdural Hematoma in Infants Associated with Meningitis Due to Bacillus Coli. DR. GEORGE D. GAMMON.

Although subdural hematoma in infants is well known, the fact that the hematoma can become spontaneously infected with bacilli of the colon group has received little attention, and no adequate review of the literature has been made. Reports of only 3 cases have

been found, in all of which the termination was fatal. In 2 of these cases the clinical picture was that of a child, abnormal from birth, in whom fatal meningitis developed several months later. The same sequence was present in the case of the successfully treated patient here described, although the preexisting abnormality had not previously been recognized.

Two cases of the condition are described in this report, in 1 of which the patient died; in the other case the correct diagnosis was made, and the child was successfully treated, being the first patient on record to survive. The child, a girl aged 8 months, was admitted with meningitis and septicemia due to infection with Escherichia acidi lactici. A preexisting difficulty was suspected because of the enlarged head and delay in development. Subdural taps revealed large quantities of yellow, infected fluid over the convexities of the brain. After trephination and drainage the child recovered and, during the next three years, developed into a bright girl, with only slight residual hemiparesis.

In the second case the patient, aged 5 days, died of meningitis due to the colon bacillus and a large subdural hematoma in the middle fossa. Cultures of blood taken before and after death yielded the bacilli, and at autopsy the same organism was obtained from the lungs, the middle ear and the meninges.

In addition to these cases, 3 instances of meningitis due to Bacillus coli are discussed, in 1 of which infection occurred through a congenital sinus leading from the skin to the cisterna magna, a kind of rudimentary meningocele. Death was due to medullary abscess. In another case block of the ventricular outflow developed, the ventricular fluid remaining infected while the lumbar fluid became sterile. Intraventricular injections of sulfathiazole finally sterilized the ventricular fluid but did not prevent death, several months later, of obstructive hydrocephalus. In a third case death was due to intraventricular abscess.

Infection of the blood stream with the colon bacillus probably occurs in the neonatal period, and, in the presence of subdural hemorrhage, the organism may invade the fluid and only later spread to produce meningitis. Drainage of the infected hematoma is essential to recovery. Delayed development and enlargement of the head in infants with meningitis should arouse suspicion of an underlying hematoma. The colon bacillus is not the only organism which may produce infection in cases of subdural hematoma; the pneumococcus, meningococcus, streptococcus and Bacillus suipestifer have also been reported as responsible agents.

DISCUSSION

DR. J. ALBRIGHT JONES: I have not yet gone over enough charts to report in detail on cases of meningitis in general. In a rather large series collected by Davison, the various types of meningitis were arranged in order of frequency of occurrence. Leading the list was meningococcic meningitis, in 30 to 40 per cent of cases; then followed tuberculous meningitis, in 31 to 32 per cent; influenzal meningitis, in 4.5 to 14 per cent; streptococcic meningitis, in 7.5 to 20 per cent; pneumococcic meningitis, in 7.5 to 17 per cent, and miscellaneous types, including the form due to B. coli, about 3 to 7 per cent, the exact proportion depending on the epidemic studied.

Barron reported a series of 42 cases of meningitis in infants (19 in the newborn), with the following age distribution: 2 weeks to 1 month, 4 cases; 1 to 2 months, 7 cases; 2 to 3 months, 8 cases, and 3 to 5 months, 3 cases. Levinson collected a series of 60 cases representing the 1 to 6 month age group and tabulated the frequency of the causative organism as follows: the tubercle bacillus, 43 cases; the meningococcus, 5 cases; the pneumococcus, 3 cases; the influenza bacillus, 3 cases; the streptococcus, 2 cases; Streptococcus viridans, 1 case; Staphylococcus aureus, 1 case; Staphylococcus albus, 1 case, and B. coli, 1 case. Cases of meningitis due to B. coli are rare and involve almost exclusively the newborn; the disease is almost invariably fatal.

One who has followed the trend of treatment of meningitis during the last few years or who has gone over the charts is struck by the present low mortality rate of meningococcic meningitis and by the fairly low mortality rate of influenza meningitis. The report of Hodes and associates indicates a fairly low mortality rate for pneumococcic meningitis, which I am sure is better than most hospital records will show.

One is impressed by the importance of early diagnosis. The majority of children who recover from meningitis, particularly from the influenzal and pneumococcic forms, are those whose disease is diagnosed early and for whom administration of the proper drug was instituted immediately. A study of the old charts for comparative data shows that a few years ago, when the sulfonamide compounds first came into use, a change to another drug was often made if the effect of the sulfonamide therapy was not apparent in two days. Treatment had yet to be standardized.

There has been a good deal of discussion about the use of sulfathiazole as the proper treatment for certain bacteria. One should consider the possibility that a low sulathiazole concentration in the spinal fluid may be just as effective as a higher concentration of the other sulfonamide compounds.

With regard to the use of influenza rabbit serum, there is no doubt that patients with the influenzal type of meningitis who received rabbit serum did better than the patients with this disease who were treated in any other fashion. Sometimes the early diagnosis of meningitis is difficult. Unaccountable irritability and any suggestion of increased fulness of the fontanel in a child who was apparently well the day before should compel consideration of meningitis. Spinal puncture should be made early for diagnosis, but frequent spinal punctures are seldom indicated during treatment.

DR. WILLIAM L. WHITE: My associates and I have been particularly interested in infections with B. coli since attempting to evaluate the role that these organisms play in wound infections. It is our impression that the bacilli of the coli aerogenes group are largely saprophytic contaminants and are seldom etiologic agents in wound infection. This impression has been borne out in our experience with the use of penicillin. Wounds which contain organisms of the coli aerogenes group have been found in most instances to contain either staphylococci or streptococci on culture of material from the recesses of the wound. When wounds thus contaminated or infected are treated with penicillin, it is usual to see the gram-positive cocci disappear rather promptly, followed soon by the gram-negative bacilli.

We recently had an interesting experience in the treatment with penicillin of a child with chronic osteomyelitis of the left femur. The penicillin was being administered by continuous intravenous drip when suddenly the temperature rose to between 103 and 104 F. Cultures of the blood and of the solution of penicillin were made immediately, for the child's temperature had been normal for several weeks prior to this time. The penicillin therapy was discontinued. Twenty-four hours later the report stated that both the cultures were loaded with organisms of the B. coli group. Twelve

hours after administration of the penicillin was stopped the temperature was normal, and the child was sitting up and playing in his crib. Here was an example of simple intravenous inoculation with a pure culture of an organism of the B. coli group. Without any form of specific therapy the child made a prompt recovery.

With regard to B. coli meningitis, I have often wondered whether there is not an underlying necrotizing organism which fails to grow under the usual bacteriologic methods available. Certainly, B. coli in the body cavities will produce toxemia, due to the absorption of bacterial end products, and is capable of producing a localized pyogenic response. It seems doubtful whether B. coli possesses the power of invasion and whether it deserves all the blame it has received.

DR. GEORGE D. GAMMON: From my reading of the literature it is obvious that the bacteriologic features of the gram-negative colon group are not entirely clear and their identification is far from complete. Through the literature the hope is expressed that subsequent or further subdivision of this group of organisms will explain the innocence of some of the types and the pathogenicity of others.

Dr. White has reflected the opinion often stated in the medical literature, namely, that the presence of the colon bacillus in the blood stream is frequent and means nothing; yet in the cases reported children died of obvious lesions from which only this organism could be recovered. In the literature cases in which organisms were associated with the B. coli group are far fewer than are those in which pure cultures of B. coli were obtained. If another organism was present, either it was not grown out or it was a virus.

Encephalitis Due to Toxoplasm. DR. GABRIEL A. SCHWARZ and DR. JAMES WENDELL.

Out of the cases of uncertain organic disease of the brain occurring in infants and children, the work of Wolf, Cowan and Paige has established a clinically recognizable entity due to invasion of the central nervous system by the protozoon Toxoplasma. We present 2 clinical cases of toxoplasm encephalitis of the so-called latent infantile type.

Both patients were white girls 5 years of age. The first child had severe pneumonia at the age of 9 weeks, after which most of her symptoms were first noted. The other child had an illness at birth involving the respiratory tract and congenital dislocation of the hips. Both patients had had convulsive seizures earlier in childhood. The clinical observations were essentially similar. Both patients showed mental deficiency. Visual acuity was greatly reduced, and there was ocular nystagmus. Ophthalmic examination revealed white nerve heads and chorioretinitis, involving particularly the macular areas. Pneumoencephalograms revealed symmetric dilatation of the ventricular systems. Roentgenograms of the skull in each case showed multiple irregular calcifications distributed throughout both cerebral hemispheres, particularly in the occipitoparietal region. A specimen of the blood of the first patient, examined by Dr. Abner Wolf, contained antibodies for Toxoplasm.

An effort was made to establish the clinical features of human toxoplasmosis on the basis of the reported cases and our own experience.

DISCUSSION

DR. HENRY WYCIS: It is important that Dr. Schwarz has outlined three forms of this disease. The adult form is especially important to bear in mind. Recently, 2 cases of the adult form were reported in South America. Symptoms referable to the lungs and signs of meningitis were the predominant features. The possibility that carriers of this disease may exist has been borne out in recent experimental work showing that mice given injections of the Sabin strain of Toxoplasma may recover if the dose is not too great. These mice may harbor the infection for a long period and appear to be cured. However, emulsions of the brains of these mice, inoculated into another animal, may produce an acute illness, which may be fatal. Two slides are shown of a case of toxoplasmosis in a 14 month old Negro infant whom I studied. The encephalogram revealed communicating hydrocephalus with ventricular distortion; multiple tiny calcifications were scattered throughout the brain. This infant had convulsions, hemiplegia of the left side and blindness. There was focal chorioretinitis. Studies of the serum gave positive results.

DR. MATTHEW T. MOORE: Cases of toxoplasmosis of the infantile and juvenile forms are rare. However, when convulsive seizures occur early in infancy and there are the associated signs of pronounced choreoretinitis, the appearance of which is thought to be characteristic, and roentgenographic evidence of minute calcifications diffusely distributed throughout the brain, the condition may be considered in the category of toxoplasmosis. Dr. Winkelman and I are now studying the brain of an infant who lived three days after a cesarean delivery. The mother, aged 28, had diabetes mellitus and was delivered at full term. This child presented the cytologic picture of the blood characteristic of erythroblastosis fetalis, similar to the condition in the case described by Steiner and Kaump (*J. Neuropath. & Exper. Neurol.* **3**:36 [Jan.] 1944). The patient had numerous convulsive seizures and showed pronounced jaundice and fine petechial hemorrhages in the skin.

The brain showed a moderate degree of internal hydrocephalus. Section revealed numerous lesions, distributed mainly in the vicinity of the basal nuclei and the pulvinar, and a few in the parietal lobes, which grossly appeared granulomatous. The histologic appearance of these lesions was that of a granuloma, perhaps several weeks or more old, which contained calcium deposits, as verified by the Kossa stain. It is interesting that the calcium in these lesions was diffusely distributed and was of an amorphous, rather than a crystalline, composition. I dare say that if roentgenograms of the head had been taken, these calcifications would have been seen. In accordance with the views expressed in the literature, we feel that this condition is antenatal and that the juvenile form is seen simply because the child succeeds in living beyond infancy despite the cerebral lesions. In our case we observed the toxoplasms in cysts and scattered in the brain tissue.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

PAUL I. YAKOVLEV, M. D., *Presiding*

Regular Meeting, Feb. 17, 1944

Psychiatry in a Naval Prison. COMMANDER THOMAS HOUSTON PARGEN (MC), U. S. N. R.

Rehabilitation and restoration to duty is not new in the Navy. During peacetime, since the vast majority of men were in prison for felonious crimes, which pre-

vented any possibility of their return to duty, not many were eligible for restoration; but of the offenders who qualified, 60 per cent were reclaimed. It is interesting to note the swing of a Naval prison population from the status of peacetime to that of wartime. An analysis of the population at Portsmouth, N. H., on Jan. 1, 1942 revealed that 95 per cent of the prisoners were there for felonious offenses, while only 5 per cent were confined for strictly military violations. On Jan. 1, 1943 a similar survey showed a direct reversal of these figures, namely, that only 4 per cent were in for felonious acts while 96 per cent were convicted of some violation of Navy regulations. The latter group represents a cross section of average American youths who have been quickly uprooted and thrown into an utterly strange and bewildering environment. They are not criminals; they are not even bad boys. They are of every race, color, creed and social stratum, and all states are represented. Ninety per cent of these men were in trouble for absence from duty—absence without leave, absence overleave or desertion.

At different times in the past, and for varying intervals, there have been psychiatrists attached to the Navy Yard dispensary or to the Naval hospital, with additional duty at the Naval prison. They came to the prison usually once a day to make sick calls and to examine any prisoner who had been observed to act or talk queerly. If the prisoner was found to be psychotic, he was transferred to the hospital. If not, he was recommended for discipline.

On July 31, 1942 I was sent to Portsmouth to organize and establish the psychiatric unit and to integrate it with the prison administration toward the more cohesive functioning of the rehabilitation program. Because psychiatry in military prisons is new, the psychiatrist reporting for duty to a prison, brig or detention barracks must start from "scratch." If he realizes that his duties are strictly advisory, he can accomplish much in choosing the proper approach to the commanding officer. It might be mentioned here that his success will be greater if, on arrival, he makes a careful study and analysis of the commanding officer, so that he may be able to determine which approach is most likely to succeed. Most commanding officers are of the regular line, with years of experience, and do not take well to new procedures which are radical departures from the old, accepted way of doing things.

Two important subordinates in the department are the psychiatric secretary and the classification officer. The latter at Portsouth is in charge of the interviewing staff, which consists of five enlisted men with specialist ratings, all of whom have had considerable experience in just this type of work in federal and state prisons before they entered the service. They do an excellent job of screening. Any prisoner showing the slightest peculiarity or evidence of abnormality is earmarked as "special" and sent up to the department, where he is placed on the preferred list.

The day after the prisoner is admitted, he reports to the psychiatric department. Some member of the office force fills out the information, obtained directly from the prisoner, on the face sheet; my associates and I have found this preliminary procedure of immeasurable benefit, particularly in shortening the time the psychiatrist has to spend with the prisoner. The filling out takes about ten minutes, and the material is typed in triplicate. The original is attached to the final report, which goes to the colonel; the second copy is retained for the psychiatric files, and the third copy is taken by the prisoner to the social service interviewing department. When the interview is completed, the prisoner returns to the psychiatric department with a copy of it. He then undergoes complete physical examination and neurologic examinations, a psychometric evaluation and a detailed interview to establish his mental status.

The purpose of the complete examination by the psychiatric department is (*a*) to discover and rule out nervous and mental illness; (*b*) to evaluate the personality type of the patient and his suitability for service, and (*c*) to attempt to predict his prison adjustment and the desirability of his restoration to duty and to suggest into which part of the reclamation program the prisoner will most easily fit.

None of the accepted psychiatric classifications are suited for this type of work. I have therefore outlined a classification which my colleagues and I find subserves successfully the prison objectives, particularly the purpose of restoring as many men as possible. It places all men in one of eight groups: (1) normal, (2) subnormal (lowered intelligence quotient), (3) defective (educational defects), (4) neurotic (all psychoneuroses), (5) psychotic, (6) alcoholic, (7) constitutional psychopathic types and (8) men with other abnormalities, such as epilepsy and narcolepsy.

It is readily seen that at the very beginning three of the aforementioned groups are suitable for reclamation, namely, the normal, the men with educational defects and the men with certain types of constitutional psychopathic states.

When a man who is getting full benefit of the rehabilitation program has served one third of his time, he is entitled to appear before the advisory board, which consists of eight officers, with the senior psychiatrist as chairman. All members of the board direct certain activities in the program. The prisoner's case is viewed in its entirety, and certain recommendations are made to the commanding officer, who passes the favorable ones on to the Judge Advocate General's Department, in Washington, D. C. When approval is received, the man is sent to his new station.

This system has now been functioning for a year and a half. It has gradually grown and smoothed out, so that it is proving successful. During the six month period from Aug. 1, 1943 to Feb. 1, 1944, of the total number of men who left the prison, 86.5 per cent were restored to duty. To date, only 1.03 per cent of the restored men have been unfortunate enough to get into trouble again.

DISCUSSION

CAPT. A. WARREN STEARNS: Since Commander Pargen has made his discussion entirely impersonal, I hope I shall be pardoned if I make my comments entirely personal. For many years I have been interested in the introduction of case work in the problem of prison management. I have always known that somewhere it might be possible to have the cooperation of an intelligent administrative officer and an intelligent psychiatrist. With such a condition the outlook of the prison administration might be much happier. I knew that there was a prison at Portsmouth, and I had heard that there was a psychiatrist there. Six months or so ago I visited that institution and was amazed at the spirit which prevailed. I found the morale high, with every one cooperating. Each prisoner was doing his work with great enthusiasm and was trying to excel. There was a spirit of keen competition between the wards and the various departments.

I saw whaleboats manned by prisoners, with the crews struggling for perfection, but I was most amazed when I saw antiaircraft guns in charge of prisoners. After

an extensive trip through the establishment, I was returned to the main office, and there I found the secret of the whole situation. A Marine colonel, with forty years of service, was in charge. He had visited all the leading institutions of the country and was thoroughly informed in all matters relating to prison management. There was nothing revolutionary in his attitude, just a rebellion against the inflexible ills of the old system and a willingness to accept modern ideas and to put them into operation.

Next I found Dr. Pargen. These two men, the colonel and the psychiatrist, were administering this excellent prison. So I came away reassured. For once in my life I had seen proper integration of psychiatry in prison management. The goal of every prisoner seemed to be to perfect himself.

I wanted this society to know what fine work was being done at Portsmouth; so I persuaded Dr. Pargen to come here and tell his story. It seemed important that all should know about this undertaking and so have a feeling of satisfaction in the Navy. Most important is the effect this understanding will have in teaching other administrators what can be done. While the situation at Portsmouth is ideal, the same principles could be applied to any prison, and I look forward to seeing this spirit prevail in other prisons after the war. Thus the spirit of revolt among prisoners can be set aside in favor of high morale.

DR. HARRY C. SOLOMON: The story that both Commander Pargen and Captain Stearns tell of this more or less utopian matter is heartening. I should like to ask whether the arrangement is unique. Perhaps Colonel Bloomberg could say whether the Army has anything like it. There are many points in Commander Pargen's statements which I should like to have him discuss in more detail. His statement that the difficulty with the men had arisen because of the lack of moral training of the youths of the country in the last twenty years is of extreme interest. This lack of training has been one of the most significant problems in relation to morale. The psychiatrist should have a good deal to do with the whole educational system, as well as with the parents. The second question arises from Commander Pargen's statement that men with convictions for felony are irreclaimable. Finally, I should like to have a fuller description of the building of morale. What is done? Are the men given recreational facilities and the like?

LT. COL. WILFRED BLOOMBERG: I am grateful to have the opportunity of hearing Dr. Pargen's paper because, as most here know, I have been interested in prisons and prison work, and it is remarkable to find a psychiatrist and a prison administrator working so well together. I still think we psychiatrists were responsible twenty-five years ago for setting back the cause of psychiatry in prison work by claiming too much and thinking we could solve all the problems. We are now getting to the point where we can really see what we have to offer.

I cannot state in detail what the Army is doing. I was concerned only with what the Army did in this area when it started the rehabilitation program. I can outline the plan as it existed at that time.

In the first place, the Army's program is vastly greater than the Navy's just because of the greater increase in man power of the Army than of the Navy. Consequently, the problem was broken down into sections, each service command setting up its own center for the kind of charges spoken of, such as absence without leave and desertion. I am not clear as to what type of offenders Commander Pargen is treating at Portsmouth. In the Army first offenders are not sent to a prison of this type. They are taken care of by guardhouse incarceration. I know a psychiatrist who was commissioned at the beginning of the program and is now on duty at Fort Devens, Mass. There are plans for shop work insofar as they have equipment there. There is also an honor company, with privileges accordingly, which gets as near the uniformed soldier as possible. When a man is thought fit to go back to duty, he may be returned without such limitation to a minimum fraction of the sentence as has been mentioned for the Navy. I believe no such ideal situation prevails between the psychiatrist and the administration as exists in Portsmouth. The problems of the Army, however, are about the same as those of the Navy, and I trust that a similar program will be followed.

DR. PAUL I. YAKOVLEV, Waltham, Mass.: I should like to ask what length of time is necessary for release of a military offender. Is it a matter of two to three months or a year or more?

COMDR. THOMAS HOUSTON PARGEN: In answer to Dr. Yakovlev's question, to Portsmouth come only the general courtmartial prisoners. However, in the past six months we have received a number of men with short terms in order to relieve some of the overcrowding. The sentence, however, is usually about nine months or longer. As Colonel Bloomberg stated, the Navy has a problem similar to that of the Army; that is, the prisoners are usually second, third or fourth time offenders. First time offenders are unusual. The Army has a definite advantage in that it has more control over the time a man will remain in prison. My associates and I have been working for some time on that problem and hope to be successful in getting the Navy to send the men to us with indeterminate sentences for our discretion. If a man who is ready to go in two or three months is held longer, he will retrogress. I know of specific cases in which men got into trouble because they were held too long.

In answer to Dr. Solomon's questions, I do not mean that the men themselves are irreclaimable; the Navy states, however, that certain felonies forbid the offender's return to duty in the Navy. They are given training in a trade, however, so that they are better fitted for civilian life. We have several five year men, for example. If they behave themselves, they will be released in, perhaps, three years. These men are being trained in the printing shop, the book bindery shop, the tailoring shop, etc. In the Portsmouth prison is made all prison clothing for the Navy. All clothing for the discharged men is made there. If a man is suited to this type of work, he has three years in which to learn the trade, and at the end of that time he should be able to get a position in civilian life following this trade. He learns every phase in tailoring, from buttonholes to overcoats.

Dr. Solomon also mentioned the unstable youngster. At present I am preparing a paper on this subject. The question brings up the matter of training in youth; the home life, right after the last war; prohibition, with bathtub gin, etc. We get a great many men with more instability than one would expect. But they show remarkable avidity in the way they go in for a rehabilitation program. They are eager for it. This shows their lack of early training.

As to the recreation program, we have training classes for men going back to duty. They have four hours of training and drill, and their time is also taken up with such activities as gymnasium, judo, work with bayonets, handball, baseball, football, basketball and movies five

nights a week. A good library is open all the time. There are also correspondence courses of which they can avail themselves, with credits toward a university degree.

The Ulnar Nerve and Intrathoracic Conditions. DR. H. HALE POWERS.

To the numerous causes of multiple peripheral neuritis use of the sulfonamide compounds has recently been added. Before 1925, in the Massachusetts General Hospital, I saw several patients with ulnar neuritis which seemed secondary to an infection of the respiratory tract. The 5 patients now reported on were seen about ten years later. As some of the sulfonamide compounds are known to produce multiple neuritis, careful physicians will test the peripheral nerves and will find some cases in which the ulnar nerve alone is affected, a condition which they should not attribute to use of the sulfonamide drugs. The patients reported on presented the following intrathoracic conditions: bronchitis, pleuritis, emphysema, pneumonia and shrapnel wound with empyema, and foreign bodies in the lung and pericardium. The ulnar neuritis was unilateral in 2 patients and bilateral in 3 patients. In only 2 patients was the neuritis severe enough to produce muscular atrophy. All the patients were nervous. Four had excessive sweating of the extremities; 3 had dermographia; 1 had effort syndrome; 1 had Dupuytren's contracture of the hands and feet, and 1 had osteoarthritis. I believe that all these conditions result from irritation of the autonomic nervous system. All the patients were seen before the discovery of the sulfonamide compounds, but similar disturbances have begun to be reported and to be wrongly ascribed to use of these substances. I believe that the ulnar nerve is affected in intrathoracic conditions because it derives most of its fibers from the eighth cervical and the first dorsal roots, which are within the thorax. In the literature the only reference found to the belief that conditions of the ulnar nerve are secondary to such intrathoracic disorder occurred in an earlier paper (*J. Nerv. & Ment. Dis.* **80**:386 [Oct.] 1934).

DISCUSSION

DR. MADELAINE R. BROWN: Did Dr. Powers notice any weakness of the hands, or only sensory involvement?

DR. H. HALE POWERS: One patient had conspicuous muscular weakness. He had to use his thumbs in pushing down the keys of a cash register. One of my other patients had weakness and probably muscular atrophy.

DR. PAUL I. YAKOVLEV: Did any of Dr. Power's patients show evidence of a sympathetic pupillary syndrome on the side of the ulnar paralysis? Narrowing of the pupil, sometimes associated with vasomotor disturbances and occasionally with signs of involvement of the lower portion of the brachial plexus, occurs in cases of apical tuberculosis. This phenomenon is known to internists as Rist's sign. The presence of the sign would tend to make more precise the localization of a lesion in Dr. Power's cases.

DR. HALE POWERS: I am not familiar with Rist's sign in tuberculosis. I did not observe the Horner syndrome in my patients. Four of the patients had sweating of both hands, indicating irritation of sympathetic fibers on both sides; therefore one would not expect anisocoria, and Horner's syndrome, if present, would be bilateral and therefore difficult to demonstrate.

The patient with effort syndrome and chronic bronchitis had unilateral neuritis; there was no sweating of the hands, and I did not observe the Horner syndrome. In the report of the case (*New England J. Med.* **228**: 1021 [June] 1942), in which ulnar neuritis was, I think, wrongly attributed to use of a sulfonamide drug, Horner's syndrome was not mentioned.

In cases of the Klumpke-Dejerine syndrome, Horner's syndrome also sometimes occurs, but the Klumpke-Dejerine syndrome results from lesions of the spinal meninges or of the vertebrae, producing pressure directly on the nerve roots and the sympathetic fibers. In the Rist and Klumpke-Dejerine syndromes the phenomena are produced by direct pressure on nerve roots and fibers by the proliferation of tissue, while in the syndrome I describe intrathoracic lesions, of various kinds, at a distance from the nerve roots, are associated with loss of function of the ulnar nerve and usually with sweating of the hands, which, I think, indicates irritation of the sympathetic ganglia.

Muscular Weakness Due to a Recently Recognized Cause. DR. MADELAINE R. BROWN.

Three cases are presented in which nephritis was associated with episodes of flaccid paralysis of the extremities. The attacks of paralysis resembled episodes of familial periodic paralysis. During the attack the potassium content of the serum was low and the T wave in the electrocardiogram was low or absent. The abnormal electrocardiogram in each case was similar to that observed in cases of familial periodic paralysis and of depletion of serum potassium from other causes. Serial electrocardiograms were of value in demonstrating the nature of the paralytic attacks. The oral administration of potassium chloride was apparently of benefit in overcoming the muscular paralysis. Three slides were shown: first, the electrocardiogram before, during and after an attack of familial periodic paralysis; second, the electrocardiogram taken twenty-four hours before death from cardiac arrest of a patient with chronic nephritis, a high potassium content of the serum (8.5 milliequivalents per liter), flaccid quadriplegia and a high T wave in the electrocardiogram, and, third, the electrocardiograms of 1 of the 3 patients whose cases are described in this paper taken during and after an attack of muscular paralysis.

This paper was published in *The Journal of the American Medical Association* (Brown, M. R.; Currens, J. H., and Marchand, J. F.: Muscular Paralysis and Electrocardiographic Abnormalities Resulting from Potassium Loss in Chronic Nephritis, **124**:545 [Feb. 26] 1944).

DICUSSION

DR. HENRY R. VIETS: All neurologists are puzzled by cases of periodic paralysis. The hereditary type, familial periodic paralysis, as studied particularly by Talbott (*Medicine* **20**:85-143 [Feb.] 1941), is constantly associated during the attacks with a decrease in the potassium concentration in the serum. The potassium migrates from the serum into the intracellular spaces, and the symptoms subside when the concentration of potassium in the serum is restored. Talbott stated that it is quite possible that many persons are susceptible to periodic paralysis under certain circumstances. These views are widely accepted and are sound.

Familial periodic paralysis is a rare disease. There are, however, at least two well studied families with this disease in Boston. Their histories are known for three or four generations. I have investigated two generations, and the late E. W. Taylor studied some of the earlier members of the same family. All affected persons in this area of whom I know are collaterals of these two families.

The significance of certain changes in the electrocardiogram associated with a low potassium concentration of the serum has only recently been recognized. Dr. Brown has described for the first time the occurrence of this state as a syndrome in patients with periodic attacks of muscular weakness, particularly persons who also have nephritis. This is a new and important piece of work. Dr. Brown has put parts of a puzzle together, and one is greatly indebted to her for doing so. Most important, of course, is her description of the syndrome itself. She points out, in addition, that from a study of the electrocardiogram one can deduce, without complicated studies of the serum potassium, that a patient is low in potassium. This simple test, if its dependability is upheld, is an important advance in clinical neurology.

DR. PAUL I. YAKOVLEV: I should like to ask Dr. Brown to recapitulate the essential points which are related to familial periodic paralysis. What are the reasons for the low concentrations of potassium in the blood which she finds? Is the condition in her cases a renal disorder due to loss of potassium, and familial periodic paralysis a disease of more continuous course?

DR. MADELAINE R. BROWN: I think that clinically the muscular weakness in these two conditions is the same because with both conditions there is a periodic fall in serum potassium, but the mechanism causing the decrease differs. No one understands the cause of the low potassium level of the serum in familial periodic paralysis. It is known that there is no increase in excretion of potassium before or during the attacks and that the potassium must leave the serum and enter the cells of the body. The explanation of the renal disorder is that the patient with the tubular disease characteristic of early nephritis cannot form ammonia. Instead, the kidney excretes serum bases in combination with the acid radicals.

DR. HENRY R. VIETS: Is it true that nephritis or other renal disorder has never been observed in association with familial periodic paralysis?

DR. MADELAINE R. BROWN: There is no evidence of renal disease in familial periodic paralysis.

Book Reviews

Segmental Neuralgia in Painful Syndromes. By Bernard Judovich and William Bates. Pp. 313. Philadelphia: F. A. Davis Company, 1944.

This book deals with segmental pain and tenderness, or segmental neuralgia, as it is manifested throughout the body. According to the authors, "segmental pain and tenderness, or segmental neuralgia, is defined as an area of spontaneous pain within one or more tender skin sensory segments (dermatomes)," which "in the majority of cases . . . is due to the irritation of the roots and/or nerve trunks." Treatment depends on the precipitating factor and on proper localization of the painful zone. In the acute stage treatment is directed to the specific, etiologic factors, and in the chronic stage it consists mainly of the removal of foci of infection, postural correction, use of mechanical supports, physical therapy and nerve block.

Poor body posture, its relation to segmental neuralgia in the thorax, abdomen and extremities, and its treatment, is discussed at length throughout the book. Pain originating in the brachial plexus and various syndromes resulting from spasm or hypertrophy of the scalenus anticus muscle are well described. There is a discussion of the differential diagnosis of segmental neuralgia and pain caused by visceral lesions, which is important in the avoidance of unnecessary, and often harmful, surgical operations. Pain in the lower part of the back and sciatic pain are considered. There is a chapter on the use of pitcher plant distillate and the ammonium salts for relief of pain, as well as a discussion of the treatment of other painful syndromes, including herpes zoster and occipital and trigeminal neuralgia. The control of pain arising from malignant tumor by the intrathecal administration of alcohol and ammonium salts and by radiation therapy is also considered. The book ends with a description of the technic of infiltration of nerves.

This monograph contains much excellent material, the proper application of which is undoubtedly of great value in the treatment of various syndromes whose chief manifestation is pain accompanied by tenderness. Particularly striking are the excellent results obtained by the simple procedures necessary to correct certain mechanical defects in body posture. The book is profusely illustrated, perhaps too much so. There are a photograph of a reflex hammer resting on the tendo Achillis, to demonstrate absence or diminution of the ankle jerk, and another of a tape measure placed about the calf, to illustrate measurement of the circumference of the calf. It is unfortunate that the literary style is poor. As an example of the use of words which makes the book difficult to read, the following sentence (page 261) may be quoted. "All types of pain were injected, the majority being malignancy, arachnoiditis and tabes dorsalis." Despite these defects, the book is to be recommended to persons dealing with the problems of neuralgia.

The Neurosurgical Patient: His Problems of Diagnosis and Care. By Carl W. Rand, M.D., Clinical Professor of Neurological Surgery, University of Southern California School of Medicine, Los Angeles. Cloth. Price, $4. Pp. 576, with 121 illustrations. Springfield: Charles C Thomas, Publisher, 1944.

It is difficult to decide for what readers Dr. Rand's book is designed. The volume consists of records of neurosurgical clinics almost literally transcribed. For neurosurgeons the treatment is too condescending, glib and superficial. The student or the general practitioner will probably not take the time to go through so much material in order to extract essential data. The style is careless and the editing poor. In one place the author apologizes, only too candidly: "I should have spoken of this . . . first." One wonders why he did not. The stenographic recording of the clinics has not been sufficiently adapted to the literary medium. For quick reading, the student would do well to study the excellent illustrations, together with their full and descriptive captions.

News and Comment

PSYCHOANALYTIC AND PSYCHOSOMATIC CLINIC FOR TRAINING AND RESEARCH, COLUMBIA UNIVERSITY

A psychoanalytic and psychosomatic clinic for training and research has been established by the department of psychiatry of Columbia University. The clinic will be opened in October 1944 at the Columbia-Presbyterian Medical Center, under the supervision of Dr. Nolan D. C. Lewis, executive officer of the department of psychiatry at Columbia University and director of the New York State Psychiatric Institute and Hospital. Dr. George E. Daniels, clinical professor of psychiatry, has been appointed chief of the psychosomatic service.

The following additional appointments have been announced: Dr. Sandor Rado, clinical professor of psychiatry, director of the clinic and chief of the psychoanalytic services for inpatients and outpatients; Dr. David M. Levy, assistant clinical professor of psychiatry and chief of the psychoanalytic service for children, and Dr. Abram Kardiner, assistant clinical professor of psychiatry and chairman of the seminar on comparative analysis of cultures.

Qualified physicians who are graduates of an approved medical school and have completed an approved hospital internship of not less than one year are eligible for enrolment in the three years' course of resident graduate training in psychoanalysis and psychosomatic medicine. They will be required to undergo a psychoanalysis before admission.

The course of training includes a systematic program of lectures and seminars, clinical conferences and supervised clinical work in the psychoanalytic and psychosomatic services. It is combined with two years of resident graduate study in the other branches of psychiatry, with emphasis on the related basic medical sciences.

Candidates who meet the requirements may register for the degree of Doctor of Medical Science. On completion of an acceptable, original and previously unpublished dissertation on the laboratory or clinical aspects of the specialty, and satisfactory completion of written, oral and practical examinations in the related clinical and laboratory fields the candidate may be recommended for the degree of Doctor of Medical Science.

ANNOUNCEMENT OF FELLOWSHIPS BY THE NATIONAL COMMITTEE FOR MENTAL HYGIENE

A limited number of fellowships are being offered for training in extramural child psychiatry. Selection for these fellowships is made by the National Committee for Mental Hygiene, by which eligible applicants are to be recommended for appointment. The fellows will spend one or two years in a selected clinic, the term and plan of the fellowship to be determined by the peculiar needs of the applicant. The training is pursued according to a definite plan related to the probable future functions of these fellows. Candidates for fellowship award should have had at least a general internship and two years of psychiatric service in an approved hospital for mental disease, in addition to other qualities fitting them for extramural service. Since this provision of training fellowships comes in response to a paucity of personnel in the field of mental hygiene, peculiarities of the demand are considered in making appointments. The stipends vary slightly with the location and status of the fellow, but in general they range from $2,000 to $2,400.

Requests for further information about these fellowships, and applications therefor, should be addressed to Dr. Milton E. Kirkpatrick, the National Committee for Mental Hygiene, 1790 Broadway, New York.